THE
BRITISH JOURNAL OF SURGERY

VOL. XIX. JULY, 1931. No. 73.

SOME BYGONE OPERATIONS IN SURGERY.

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V. LITHOTRITY: THE CASE OF THE EMPEROR NAPOLEON III.

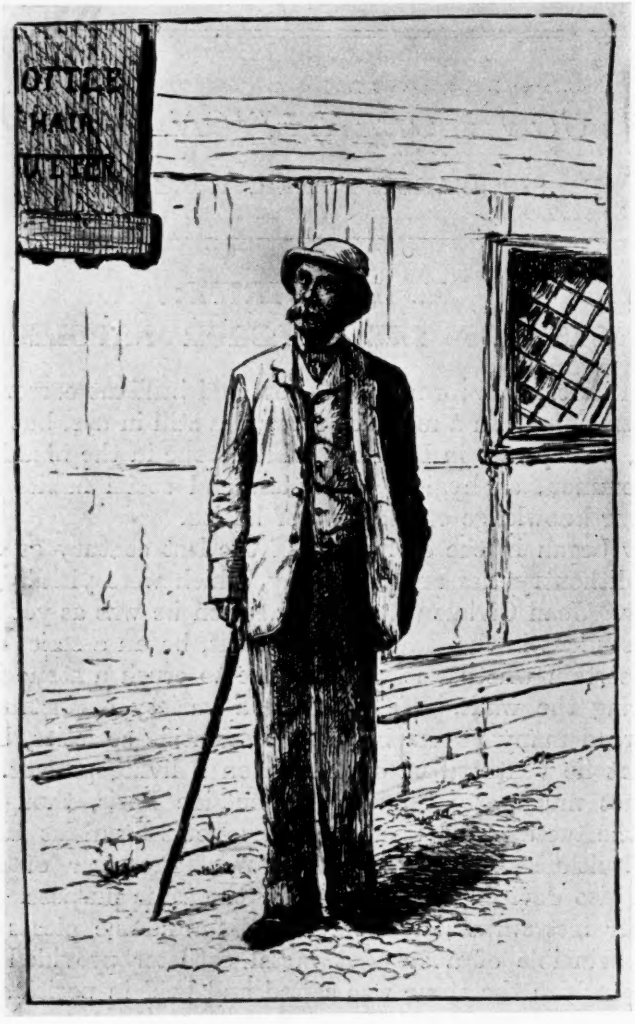
LITHOTRITY in its original form had a short and brilliant career in the history of surgical operations. In a modified form it is still in use, but with diminishing frequence, at any rate in England, where stone in the bladder is becoming yearly less common, as hygiene, dietetics, and social habits improve, with advances in our knowledge of the laws of health.

Lithotrity began in the early part of the last century as a counterblast to the use of lithontryptics and lithotomy, which were yielding very unsatisfactory results. Jean Civiale (1792–1867), when he was as yet only a second-year medical student in the University of Paris, began a series of experiments in 1817 to ascertain whether it was possible to crush a stone in the bladder without injuring the walls. He invented a variety of instruments for the purpose and read many papers before the scientific societies, but it was not until 1823 that he ventured to operate upon a living patient. His method did not attract much attention at first outside Paris, though Jean Leroy (D'Étiolles), the tactless and jealous Charles Louis Stanislas Heurteloup, and Franz von Gruithuisen, who was afterwards Professor of Astronomy at Munich, were also devising instruments for the same purpose.

The earlier instruments were large and formidable pieces of apparatus, consisting in principle of a straight metal catheter provided with a claw-shaped end by which the stone was seized and held in position whilst it was being broken up by a metal rod passed through the catheter. The stone was either pierced, drilled, crushed, or squeezed by force exerted through the rod. An attempt was made to crush the stone with these instruments at a single sitting, the fragments being allowed to pass by the natural expulsive efforts of the bladder.

The method spread slowly beyond France, and it was not until 1827 that lithotrity was undertaken in Austria and in Italy. Baron Heurteloup came to London in 1828 and lived in Vere Street until 1832. On July 24 and 30 and on August 20, 1829, he operated upon a seaman, age 64, who was under

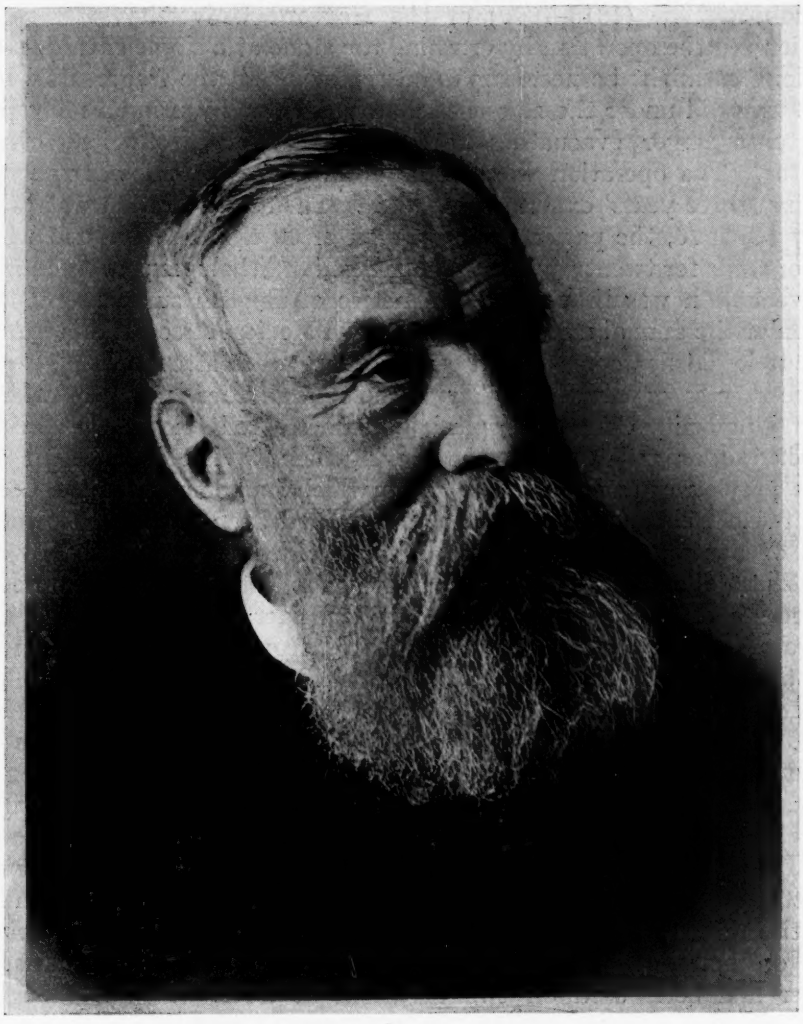
the care of Anthony White in Westminster Hospital, and this was the first case of lithotrity in England. Heurteloup had improved his technique by 1832, for he was then crushing stones at a single sitting and removing the fragments by syringing out the bladder through a catheter with a large eye. He claimed that for this purpose he invented the syringe now in general use which has three rings to enable it to be used with one hand.



SIR HENRY THOMPSON

The value of the operation was quickly recognized. Joseph Hodgson in England and Philip Crampton in Dublin performed it frequently, simplified the instrument, and improved the method of removing the fragments, though they preferred to crush the stone at repeated sittings lasting only a few minutes. The lithotrite by this time was assuming its present shape—a rod ending in a flattened blade set at an angle. A movable blade passed along the fixed one,

the sliding movement being converted into a screw motion when the stone had been seized. The female blade, at first solid, was afterwards fenestrated to allow the fragments of the stone to drop back into the bladder. The standardization of the instrument was due largely to the mechanical genius of John Weiss, the instrument maker, who then had a shop in the Strand. Credit must also be given to Charrière, of Paris, whose model Weiss improved in several particulars.



PROFESSOR H. JACOB BIGELOW

The practice of lithotrity soon fell largely into the hands of (Sir) Henry Thompson, who was devoting himself to the surgical treatment of diseases of the urinary organs. Thompson visited Paris in July, 1858, to study under Civiale, and in 1863 he published the results of his experience in the Lettsomian Lectures delivered before the Medical Society of London. Improvements had in the meantime been made in the method of evacuating the fragments of

crushed stone, and in 1846 Philip Crampton was using as a suction apparatus a glass bottle exhausted of air and connected with a catheter by means of a rubber tube provided with a stopcock. Mr. J. T. Clover, the anæsthetist, invented his apparatus in 1866, which, with slight modifications, is still in use. It consists of a rubber bulb with a glass trap to catch the fragments as they are washed out through an evacuating catheter.

The stone was crushed at repeated sittings until in 1878, when Professor H. Jacob Bigelow (1818–1890) of the Harvard University, Boston, Mass., revolutionized the operation by crushing the stone at a single sitting (continued for as long as might be necessary) and removing all the fragments whilst the patient was still under the anæsthetic. He did this by using a more powerful lithotrite, a larger evacuating catheter, and a more efficient evacuating apparatus. The operation was carefully thought out and was the result of more than three years' experimental work. His first operation was performed on May 15, 1876, the patient being a man of 60 who had suffered with symptoms of stone for twenty years. The method of litholapaxy or rapid lithotrity with evacuation was brought to the notice of English surgeons by Reginald Harrison at the annual meeting of the British Medical Association on August 8, 1878, but it did not come into use for several years afterwards.

The success of Sir Henry Thompson's lithotrities, many of which were done at University College Hospital, became so well known that in 1863 he was called upon to complete the work which Civiale had begun eighteen months previously and crush the stone which had long troubled Leopold I, King of the Belgians. The earlier sittings had been suspended owing to an attack of acute cystitis, but Thompson avoided its recurrence by using a new lithotrite on each occasion. Nine years later he was summoned to Camden Place, Chislehurst, in Kent, to consider the case of the Emperor Napoleon III. The Emperor had suffered from urinary trouble for some years, and on July 1, 1870, a consultation had been held at the Tuileries to determine the cause of his sufferings. There were present MM. Nélaton, Ricord, Fauvel, Sée, Corvisart, and Conneau, the last being his private medical attendant. Professor Sée was asked to put the findings of the consultants into writing, and Conneau undertook to obtain the signatures of the surgeons and bring the report to the notice of the Empress. The report, dated July 3, 1870, stated that the Emperor had suffered from four attacks of hæmaturia in the preceding five years and that from August, 1869, the urine had always contained pus. The shaking of a carriage and riding on horseback caused much pain. There was also dysuria, and a catheter had been used on two occasions at least. It was thought from these signs and symptoms that the Emperor undoubtedly had a renal or a vesical calculus, and exploratory measures were recommended. The report, however, miscarried owing no doubt to the turmoil prevailing in those July days just before the declaration of war against Germany. It was never signed by the consultants and it was never shown to the Empress. It seems to have been kept by Dr. Conneau, for it was seized with his other papers at the Tuileries on September 4, 1870, and was then published. The document is of historical importance owing to the date on which it was drawn up—July 3, 1870—three days before the declaration of war.

Had it been seen by the Empress and had an operation been decided upon, events might have happened very differently. The outbreak of hostilities might have been delayed or even entirely abandoned. As it was, they were undertaken by a very sick man, and in the end Napoleon came to England as an exile.

The symptoms improved for some time after the arrival of the Emperor in England from Wilhelmshöhe, but later they increased in severity until in July, 1872, Sir William Gull and Sir Henry Thompson met Dr. Conneau in consultation. The prostate was then found to be healthy, but the Emperor declined to allow a catheter to be passed. Palliative measures, therefore, could alone be recommended. The pain increased, and prevented riding, driving, and even walking, until on October 31 he consulted Sir James Paget, who advised an exploration of the bladder. This was again declined, and it was not until December 24 that Sir Henry Thompson was allowed to pass a flexible catheter, after which he impressed upon the patient the urgent necessity for an exploration of the bladder under an anæsthetic.

The necessary arrangements were made, and on Thursday, January 2, 1873, an anæsthetic was given by Mr. Clover. Sir Henry Thompson introduced a lithotrite and determined the presence of a vesical calculus, probably phosphatic, of the size of a walnut or large chestnut. The examination was made at 3.30 p.m. in the presence of Sir William Gull, Baron Corvisart, Dr. Conneau, and Mr. John Foster (brother of Sir Michael Foster the physiologist), who was then acting as Thompson's private assistant. The stone was crushed and a considerable quantity of débris was removed. The operation was followed by much pain, and a second sitting was arranged for Monday, January 6, at 10 a.m. This second sitting had to be postponed for two hours as the patient had a rigor, and it was not until 12 o'clock midday that the anæsthetic was administered, again by Mr. Clover. A large fragment of stone was then found impacted in the membranous portion of the urethra. Much delicate handling had to be undertaken before it was dislodged and further crushing was possible. There was evidence of obstruction on the day following this second sitting, but it was less complete, and it was decided not to pass any instrument. A third séance was arranged for the morning of Thursday, January 9, but by this time it is evident that the general condition of the Emperor had given rise to anxiety. He was seen at 11 o'clock on Wednesday night by his medical attendants; at 2 a.m. on Thursday by Dr. Conneau; at 4 a.m. by Baron Corvisart; at 6 a.m. by Sir Henry Thompson; and at 9.45 a.m. by all his medical attendants in consultation, as well as by Mr. Clover.

It was reported that the Emperor had slept better than on the previous night, and it was decided to proceed with a further lithotrity at 12 o'clock. He became alarmingly ill, however, at 10.25, and died at 10.45. A post-mortem examination was made by Dr. J. Burdon Sanderson, then the foremost pathologist of the day, who found extensive disease of both kidneys. The calculus weighed about $\frac{3}{4}$ oz. and measured $1\frac{1}{4} \times 1\frac{5}{16}$ in. All the other organs were healthy. About one-half of it had been crushed. Its nature is not stated, but it appears to have consisted of urates with a coating of phosphates.

Sir Henry Thompson, the operator, was a remarkable man. Born in 1820, he was the only son of the village shopkeeper at Framlingham in Suffolk, by his wife Susannah, daughter of Samuel Medley, the artist who painted the well-known portrait group of the founders of the Medical Society of London. Medley afterwards went on the Stock Exchange, made money, and was instrumental in founding University College (afterwards the London University) in 1826. Both the parents of Sir Henry Thompson were rigid nonconformists who dreaded a scientific education and disliked the idea of a profession for their son. Thompson, therefore, was employed for a short time in his father's business, but soon tiring of it, apprenticed himself to George Bottomley, a medical man practising at Croydon in Surrey. He entered as a medical student at University College, London, in October, 1844, and distinguished himself by winning the gold medals in anatomy and surgery and by his appointment under Sir John Eric Erichsen as his first house surgeon. At the end of his term of office he entered into partnership with his former master George Bottomley, but soon returned to London with the intention of practising as a surgeon. He was elected Assistant Surgeon to University College Hospital in 1853, becoming full Surgeon in 1863, Professor of Clinical Surgery in 1866, and Consulting Surgeon in 1874.

Thompson was an ardent champion of cremation as a method of disposal of the dead. He first drew attention to the subject by an article in the *Contemporary Review* in 1874, and by his energy established a Cremation Society of which he remained President until his death. A Crematorium was built at Woking, but its use was forbidden by Lord Cross, who was then Home Secretary, and it was not until March, 1885, that the first cremation was undertaken after Sir James Stephen had declared in a test case that the procedure was not illegal if it were carried out decently and without offence.

Thompson was a skilled artist, the talent no doubt being derived through his mother, though it was fostered by study under Edward Elmore, R.A. and Sir Lawrence Alma Tadema, and he exhibited frequently at the Royal Academy. He was also interested in astronomy and built an observatory at Molesey, where he had a country house. He was, too, an eminent collector of china, his collection being sold at Christie's on June 1, 1880. In the social life of London he played a great part as a host. He was celebrated for his 'Octaves'—dinners of eight persons, eight courses, at 8 o'clock. The company, the food, and the wines were of the choicest. King Edward was of the party on more than one occasion, and King George V, when Prince of Wales, was present at the 300th octave. Thompson received the honour of knighthood in 1867 and was created a baronet in 1898. He married Kate Fanny, daughter of George Loder, of Bath, and was the father of a son, Sir Henry Francis Herbert Thompson, and two daughters. Lady Thompson was well known for her musical talent as a pianist.

Sir Henry Thompson died at 35 Wimpole Street, where he had lived all his professional life, on April 18, 1904, and his body was cremated at the Golder's Green crematorium.

He wrote much, both professionally and in the lay reviews and magazines, and published two novels under the name of "Pen Oliver, F.R.C.S," the

first in 1885 being entitled *Charlie Kingston's Aunt*, the second in 1886, *All But, a Chronicle of Laxenford*, which is illustrated by twenty-four whole-page drawings by himself.

The portrait of Sir Henry Thompson is reproduced from *All But*, a copy of which was kindly lent by Mr. George Buckston Browne, F.R.C.S.Eng., who was formerly his private assistant. The original was drawn by Sir Henry Thompson, and represents him as he appeared in 1885 in Framlingham, his native village. The portrait of Professor Bigelow is from a photograph taken in 1888.

THE PRESERVATION OF THE ILEOCÆCAL SPHINCTER IN RESECTION OF THE RIGHT HALF OF THE COLON.

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REASONS FOR EXCISION OF THE ILEOCÆCAL SPHINCTER.

Resections of the colon are performed, in the great majority of cases, for the removal of malignant growths. In such an operation the growth itself, as much of the bowel above and below and of neighbouring structures as may conceivably have been involved by cancerous cells, and the whole of the lymphatic vessels and glands which drain the affected area, must be removed in one block. Since the lymphatic channels from the colon accompany the blood-vessels, and the intermediate and main lymphatic glands are grouped along them, any such radical operation implies the ligature of the corresponding colic artery shortly below its origin from the superior or inferior mesenteric trunk. Thus a second factor arises: the parts removed must include not only those necessary for eradication of all the areas which may have undergone malignant invasion, but also those whose viability has been endangered by ligature of the main artery. In the case of growths lying anywhere between the ileocæcal valve and the hepatic flexure, these considerations demand the removal of the area supplied by the ileocolic and right colic arteries, and therefore a resection which is sometimes wider than is necessary upon grounds of pathology alone.

Friedrich's operation, resection of the intestine from a point six inches above the ileocæcal valve to a point four inches below the hepatic flexure, with the peritoneum overlying the ileocolic and right colic arteries and the lymphatics accompanying them, has for many years been accepted as an eminently satisfactory procedure for the treatment of operable cancer in the right half of the colon. The immediate and late results of Friedrich's operation as regards safety and prospects of cure are admirable, but it implies the removal of one of the most important pieces of mechanism in the physiology of digestion, the ileocæcal sphincter. In cancer surgery this is unavoidable, for the need for eradication of the disease overrules all other considerations, and cancer surgery has hitherto been dominant. Of late, however, operations upon the colon for the treatment of non-malignant conditions have come to assume an increasing importance. Diverticulitis, stasis in the proximal colon, and dysfunction after suppurative appendicitis, may all demand resections of the right half of the colon in which any wide removal of the lymphatic drainage area is unnecessary. The purpose of this paper is to suggest that in such operations the physiology of the ileocæcal angle should receive greater consideration.

THE STRUCTURE, MOVEMENTS, AND FUNCTION OF THE ILEOCÆCAL SPHINCTER.

The observations of Keith in 1903 and of Rutherford in 1914 have established the fact that the ileocæcal opening is a muscular sphincter, which does not depend in its action upon any valvular mechanism. In life the termination of the ileum is seen as a hemispherical papilla about 1·8 cm. in diameter and projecting about 1 cm. above the surrounding cæcal wall: it is smooth and scarlet, in contrast to the pink and folded cæcal mucosa. The frænula described in anatomical text-books are never seen. The sphincter is formed by the fusion of the circular and longitudinal fibres of the termination of the ileum and those of the adjacent cæcum; in addition some independent circular fibres are grouped round its summit. The ileocæcal sphincter derives its nerve-supply from the sympathetic system. Section of the splanchnic nerves is followed by permanent relaxation; on the other hand, direct stimulation of these nerves, or indirect stimulation by adrenalin, produces violent contraction of the summit of the sphincter, while the movements of the rest of the intestine are inhibited. Stimulation of the vagi or pelvic nerves has no effect. Keith has demonstrated that the nodal tissue forming Auerbach's plexus is especially developed at the level of the ileocæcal sphincter, as at the cardia.

Rutherford has described the movements of the ileocæcal sphincter, as observed through a cæcostomy opening. During periods of activity, waves of peristalsis can be seen through the relaxed cæcal wall progressing along the terminal ileum at regular intervals. When the wave of contraction reaches the cæcum, the sphincter relaxes, and the papilla becomes wider and less prominent. About 4 c.c. of liquid fæces and gas enter the cæcum, and the sphincter then shuts and becomes firmer and less prominent.

The chief function of the ileocæcal sphincter is to prevent the ileal contents passing too rapidly into the cæcum. Hurst has shown that a barium meal reaches the end of the ileum one hour before any appreciable quantity enters the cæcum. The ileum is often full four or five hours after the last traces of a meal have left the stomach. In consequence the products of digestion collect in the terminal ileum, where they remain for a longer time than in the stomach. During this period active segmentation can be observed in the terminal four inches of the ileum, but peristalsis is always very feeble. The ileocæcal sphincter has therefore the same effect on the contents of the terminal ileum as the pyloric sphincter has on the contents of the stomach, controlling the outflow, so that sufficient time may elapse for the complete digestion and absorption of foodstuffs. This physiological ileal stasis is so well regulated that the chyme which enters the cæcum only contains very small quantities of nutritive material in solution.

The ileocæcal sphincter relaxes whenever a wave of peristalsis passes over the terminal coils of the ileum. Though these waves start shortly after the arrival of the chyme at the ileocæcal angle, they are at first very infrequent. As a result of the ileocæcal reflex described by Hurst, active peristalsis takes place at the lower end of the ileum each time that a meal enters the stomach; the sphincter relaxes, and chyme enters the cæcum. The sphincter is not

called upon under normal conditions to prevent reflux of fæcal matter into the ileum, since antiperistalsis does not occur in the proximal colon unless there is some distal obstruction. When such movements occur, the sphincter cannot withstand any degree of increased pressure on its colic surface.

Spasm and Incompetence of the Ileocæcal Sphincter.—Hurst believes that ileal stasis is rarely encountered except under conditions of organic obstruction at the ileocæcal angle. When true ileal stasis is seen in the absence of organic obstruction it is usually due to achalasia of the ileocæcal sphincter determined by a chronically infected appendix. This condition is exactly comparable to that of pyloric achalasia in duodenal ulcer.

Incompetence of the sphincter does not occur as a clinically observed phenomenon. After ileocolostomy with or without resection of the terminal ileum, the controlling action of the ileocæcal sphincter is entirely abolished and the contents of the ileum pour straight into the colon. The lower four feet of the small intestine become dilated, their muscular coats hypertrophy, and their lumen contains semi-solid fæcal matter. The terminal stages of intestinal digestion and absorption are cut short, and much of the nutritive material in the chyme is lost to the body. Much more serious is the constant presence of the putrefactive bacteria in the small intestine, where they find abundant material on which to multiply, and where the free blood-supply and specialized absorptive function of the mucous membrane facilitates the entry into the circulation of the toxins which they produce.

The resection or exclusion of the ileocæcal sphincter for intestinal stasis in young patients is fraught, in the majority of cases, with disastrous consequences. At first, if constipation was very marked before the operation, the patients may feel much better, and this improvement may last for as long as five years. Later, progressive ill health, suggesting absorption of toxins, sets in. Irritability, nervousness, insomnia, a feeling of extreme weakness and tiredness, headaches, nausea, loss of appetite, and loss of weight are the main symptoms. No treatment has much effect in alleviating these troubles.

THE OPERATION OF TRANSPLANTATION OF THE ILEOCÆCAL SPHINCTER.

Any modification in technique which enables the essential object of an older operation to be attained, and at the same time preserves to a greater extent the function of the part, is a step forward. The operation of transplantation of the ileocæcal sphincter here described is an example of such an attempt to retain function. The immediate results of the operation in the two cases in which it has been carried out have been so satisfactory that they justify the publication of a method which has only had such brief trial. The principle of the operation was suggested by Dr. A. F. Hurst, who had both patients under his care in New Lodge Clinic. Full clinical details of the condition for which the operation was performed are appended at the end of this paper. The actual procedure was the same in each case.

The abdomen is opened by a right paramedian incision, 6 or 7 in. long,

and about an inch from the mid-line, the rectus being displaced outwards. After separation of adhesions, and a general survey of the abdominal viscera, the peritoneum is divided to the outer side of the ascending colon (*Fig.* 1). This division is made at the edge of the bowel, or slightly overlapping it, so that plenty of peritoneum shall be available at the end of the operation for covering the whole posterior abdominal wall, and is carried upwards round the hepatic flexure for 2 in. along its superior border, and downwards round the attachment of the cæcum in the right iliac fossa, to continue for 1 in. along the inferior aspect of the attachment of the mesentery of the ileum. The cæcum, ascending colon, and hepatic flexure are mobilized by stripping them inwards as in the first stages of Friedrich's operation. In this conservative operation, however, the mobilization does not extend inwards to the

FIG. 1.—Division of peritoneum and mobilization of colon.

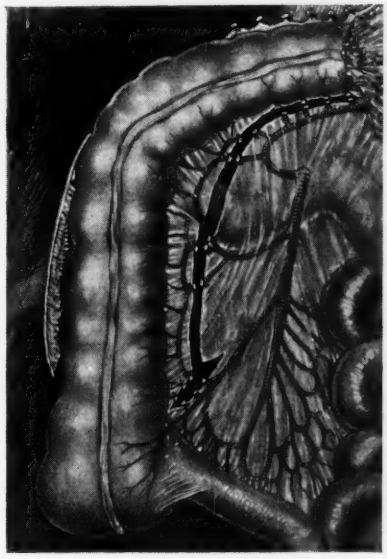

FIG. 2.—Division of vessels and isolation of ileocolic bundle.

ureter, and the stripping is less vigorous, the retroperitoneal layer of fascia being left on the muscles.

The mobilized colon is now held up, and the ileocolic artery and its branches are identified through the translucid peritoneum : the branches going to the right are divided between double ligatures about half an inch from the main ileocolic artery (*Fig.* 2), and the peritoneum is incised along the line of this division from the inner margin of the cæcum for about 1½ in. mesially. The blood-supply to the ileocæcal angle, and the accompanying sympathetic plexus which innervates the sphincter, are thus left intact. Division of the peritoneum is carried upwards on the inner side of the ascending colon at a distance of about an inch from its wall, the right colic artery or its branches being cut between double ligatures. A point from one-third to one-half the distance along the transverse colon from the hepatic flexure is now selected for division, such a length being left as will allow the blind proximal end of

the divided colon to lie comfortably at the old site of the hepatic flexure without tension, yet without looping of the remaining transverse colon. The branches of the middle colic are divided close to the wall of the colon for a distance of 1 in. on each side of the selected spot, and the attachments of the great omentum to the superior border of the part of the colon to be removed are tied off close to the bowel and divided. The colon is crushed with a powerful clamp 1 in. wide (*Fig.* 3), and tied in the crushed portion by two ligatures, $\frac{1}{4}$ in. apart, which are left long. Proximal and distal to these ligatures, two purse-string sutures are placed round the bowel but not tied (*Fig.* 4). The colon is now divided between the ligatures with a cautery, the ends are held up by an assistant, and invaginated as the purse-string sutures are tightened by forceps which are immediately put aside. The blind end of the distal colon is further closed by a second purse-string suture.

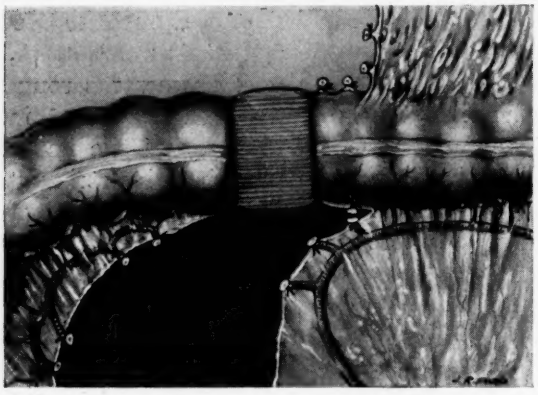

FIG. 3.—Transverse colon crushed at point selected for division.

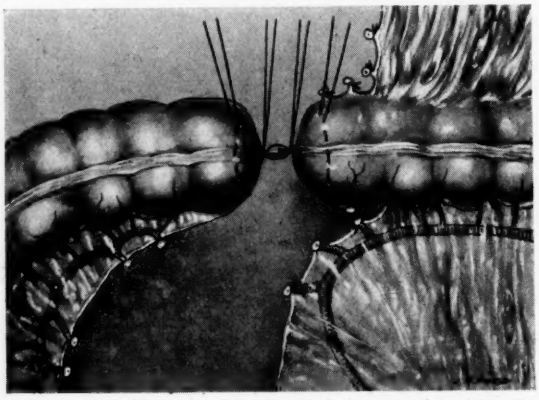

FIG. 4.—Transverse colon ligatured in crushed portion: two purse-string sutures in position, ready for tying.

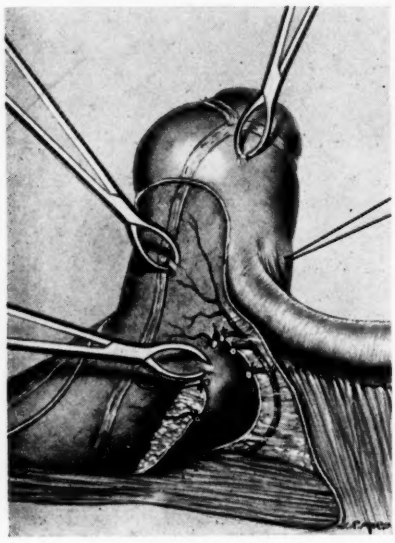

FIG. 5.—Diamond-shaped area on the medial cæcal wall marked out for transplantation. (In the figure the ascending colon is rotated clockwise through 120° to bring the ileocæcal region towards the observer.)

The cæcum is now prepared for anastomosis. The posterior wall has no peritoneal coat, but is covered with loose fat, in which the cæcal vessels run before they pierce the muscular layer. All fat and connective tissue is cleared away along the line of suture, and any small vessels which are divided in the process are ligatured. A diamond-shaped piece of the medial cæcal wall (*Fig.* 5), with the ileocæcal junction

at its centre, is then marked out for transplantation. The longer diagonal of the diamond, which lies in the long axis of the bowel, is 3 in. long, the shorter, at right angles to this, is 2 in. This area is marked out by three Lane's forceps attached at its upper, lower, and posterior angles to the cæcal wall, and a stitch at its anterior angle. The transverse colon immediately beyond the closed end is now emptied of contents by gentle pressure, and a loop 5 in. long and including about half its circumference is held in an intestinal clamp so that the anterior longitudinal band lies along its summit.

FIG. 6.—Cæcum brought up to transverse colon: insertion of first row of stitches.

The ileum is controlled by a light clamp placed across it 3 in. from its termination. The cæcum is brought up to the loop of transverse colon, with its posterior surface towards the colon, and its blind end to the right (*Fig.* 6). Before the anastomosis is commenced, a strip of gauze soaked in flavine solution is laid between the two portions of bowel behind the line of junction, and abdominal mops, also wrung out in flavine, are arranged round the operation area to prevent any possible soiling.

The posterior cæcal wall is laid along the loop of colon, and the points marked by Lane's forceps are attached by three stitches of 00 catgut, the upper and lower angles of the diamond being stitched to the longitudinal band, the posterior angle $\frac{1}{2}$ in. below it. The stitch at the left-hand end of the anastomosis is left long, with the needle still on it. The cæcal wall between these stay sutures is then fixed to the transverse colon by a series of interrupted stitches about $\frac{1}{6}$ in. apart. The cæcum is now emptied by pressure, and two long Ochsner forceps are placed across it from the inner side, one above and one below the ileocæcal angle (*Fig.* 7): these forceps outline a diamond slightly larger than that marked out by the stitch and Lane's clamps, their points meeting in the middle of the cæcum. A knife is run along the ileocæcal side of these forceps, and the colon, closed

FIG. 7.—Posterior half of first row of stitches completed. Cæcum clamped off prior to removal.

at one end by a purse-string suture, and at the other by the two clamps, is removed.

The exposed surface of the cæcum and ileocæcal valve is rapidly mopped dry and swabbed with gauze soaked in flavine solution. The transverse colon is opened by an incision along the longitudinal band, and its interior also mopped clean and swabbed with flavine (*Fig.* 8). A stitch of 00 catgut is next inserted at the right-hand corner of the anastomosis, the needle piercing the transverse colon from the mucosa outwards and re-entering the cæcum from without inwards; this stitch is tied on the mucosa and left long. A second stitch, piercing all the coats of the adjacent edges of transverse colon and cæcum, is inserted half-way along the line of anastomosis, tied, and left long. The continuous hæmostatic suture is then started at the left-hand end of the opening. This suture is of 00 catgut with a needle at each end, and is inserted in the same way as that at the right-hand corner, the knot being tied on the mucosa at the centre of the catgut. It is continued as an over-and-over stitch through all the layers, the first and second stay sutures, when reached, being knotted to the continuous one, and cut short. The return layer of the hæmostatic suture is done by a simple over-and-over stitch with pull on the mucosa which I learned five years ago from Tyrrell-Gray, and have since used for all anastomoses (*Fig.* 9): it inverts the mucous edges completely and includes all the vessels, being, in my opinion, preferable to the Connell, about whose efficacy for preventing leakage and controlling hæmorrhage I have always entertained the gravest doubts. My own modification is to use two needles, so that the suture line shall not finish at a corner—the weak spot of any anastomosis. The suture having reached the right-hand corner, and being tied to the stay stitch, the needle is taken straight out at the corner of the opening in the transverse colon, straight in at that on the cæcum, and

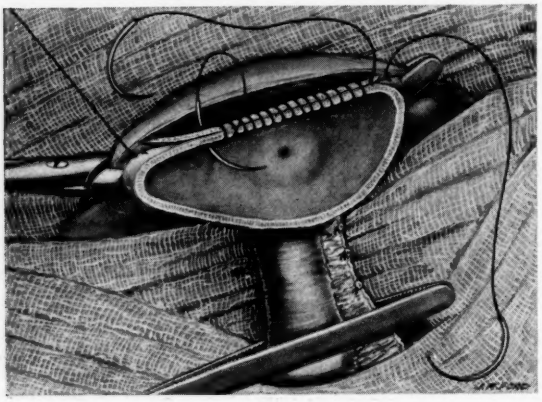

FIG. 8.—Cæcum removed, exposing ileocæcal sphincter. Insertion of first half of hæmostatic suture.

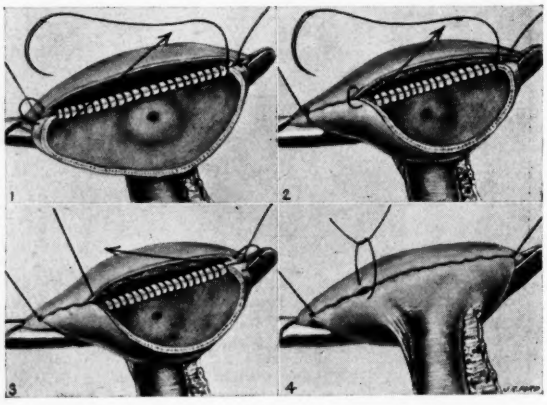

FIG. 9.—Detail of return layer of hæmostatic suture.

pulled tight on the mucosa (*Fig.* 9, 1): it is again passed from within out through the wall of the transverse colon $\frac{1}{6}$ in. beyond the corner, from without in through the cut edge of the cæcum the same distance beyond the corner, and pulled tight. Two more double stitches are taken and pulled tight on the mucosa, so that the first half-inch of the return layer is now inverted at the right-hand end (*Fig.* 9, 2). The needle is then passed from within out through the edge of the transverse colon just beyond the last stitch, and laid aside. The second needle is used to invert the left-hand corner in a similar manner (*Fig.* 9, 3). It is first passed straight out through the cæcal corner and straight in through that in the transverse colon, and pulled tight on the mucosa. The suture is continued from left to right, each stitch, as it is pulled tight from within, inverting the two edges, till the other end of the suture is reached. The needle is then passed through the cæcal wall from within outwards. The two ends, both of which appear on the outer coat, are now tied, inverting the last bit of the edge (*Fig.* 9, 4).

FIG. 10.—The operation of ileocæcal transplantation completed.

The needle which was left on the stay suture fixing the outer coat of the cæcum to the longitudinal band on the transverse colon at the left-hand end is now taken, and a continuous Lembert suture is put in from left to right, inverting the line of the hæmostatic suture; this is tied to the stay suture left at the right-hand end. A third layer, a running Lembert suture inverting the edges still further, is then inserted all round the anastomosis, which is turned round to enable its posterior surface to be reached.

The raw areas in the abdomen are now covered with peritoneum (*Fig.* 10). The gap between the upper leaf of the mesentery of the ileum and the cut edge of the transverse mesocolon has been folded on itself, and is closed by a short continuous suture. The lower leaf of the mesentery is similarly sewn to the edge of the parietal peritoneum, where it was divided on the outer side of the ascending colon. The blind end of the transverse colon is laid on the bed of the hepatic flexure, and fixed to the edges of the peritoneum on the posterior abdominal wall by a few interrupted sutures. The cut edge of the omentum, if redundant, is fixed by one or two stitches over the anastomosis.

The whole area of operation is lightly mopped with flavine solution, and the abdomen closed.

DISCUSSION.

The operation here described is difficult and severe, and by no means without risk. It is, however, so essentially sound in principle, and has been so successful in the two instances recorded below, that I intend to perform

it in further cases where the disability justifies a procedure of such magnitude. One difficulty is that the patients are wasted, toxic, and discouraged, and already in poor condition to withstand a severe ordeal. Extensive adhesions from previous operations must be separated, a time-consuming task, before resection can be commenced.

In the operation itself the chief difficulty is that of making a secure and leak-proof transplant into the side of the colon. Care is required in cutting the cæcal patch to ensure that its size is conformable to that of the opening in the colon, and that the ileal opening is at its centre and not near enough to any edge to suffer damage to its musculature or nerve-supply. The suture of the posterior cæcal wall to the transverse colon is the least secure part of the anastomosis, since the whole of this wall presents a rather indefinite surface uncovered by peritoneum, an enlarged 'danger area'. For this reason I have used interrupted stitches for the posterior half of the first layer, and have reinforced the whole anastomosis with a third layer of sutures.

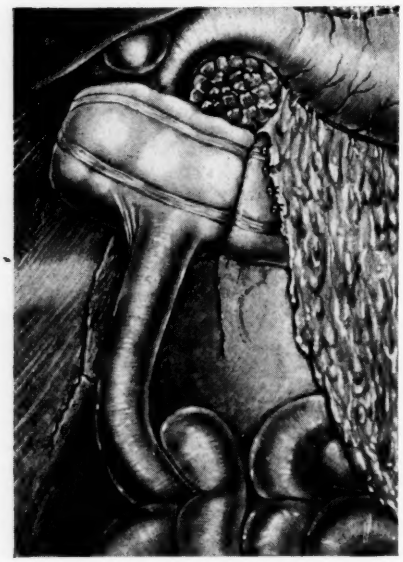

FIG. 11.—The operation of cæcal transplantation.

After operation there remains the further difficulty of getting the bowels open without putting any strain on the new stoma. To minimize this strain, I have had the patients in hospital for a week before operation, and given them 4 oz. of Petrolagar daily. On the morning of the operation the colon is washed out. After operation Petrolagar is again given every four hours. In spite of these precautions, it has been necessary in each case to give a small glycerin enema before the bowels were evacuated.

On account of these difficulties and dangers, I performed in a third case the operation of transplantation of the cæcum. The first stages of this operation were identical with those described. An end-to-end anastomosis was then made, by the aseptic method of Pringle and Schoemaker, between the cæcum, half an inch beyond the sphincter, and a point in the transverse colon about its middle, chosen to allow the cæcum to lie naturally in the bed of the hepatic flexure after anastomosis (*Fig.* 11). I have found that the aseptic method has robbed end-to-end anastomosis in the colon of its difficulties, not because it is aseptic, but because the two ends of gut are held straight and of identical length till the suture line is complete. The junction, when finished, is a much neater piece of work than I have ever succeeded in producing by the open method. Pringle's forceps have failed me, and Schoemaker's I have found too short; those I use are made by Down Bros., and are Schoemaker's with blades 1 cm. longer than the original pattern. In this operation the result after three months is identical with that following the operation of ileocæcal transplantation, but the radiograms taken three

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weeks after operation show a very definite diaphragm at the suture line, leading to marked stasis in the terminal ileum.

The operation of transplantation of the cæcum is shorter, easier, and probably safer, than that of transplantation of the ileocæcal valve. When resection of the ascending colon is required for non-malignant conditions in which the cæcum is undamaged, as in the case reported, it is probably the better; but when, as in most cases following previous operations, the cæcum is damaged, the operation of transplanting the valve alone into a new cæcum made from colon which is still healthy, is more likely to give results that are permanently satisfactory.

CASE REPORTS.

Case 1.—**Female, age 37. Transplantation of the ileocæcal sphincter.**

HISTORY.—Appendicitis in 1922; typhoid fever in 1924; tonsillectomy in 1927. Laparotomy in 1928 for pyrexia with pelvic symptoms, supposed to be due to salpingitis; only a small cyst of the right ovary was found, and this was removed. Since the last operation, the patient had never been well, and had to abandon her

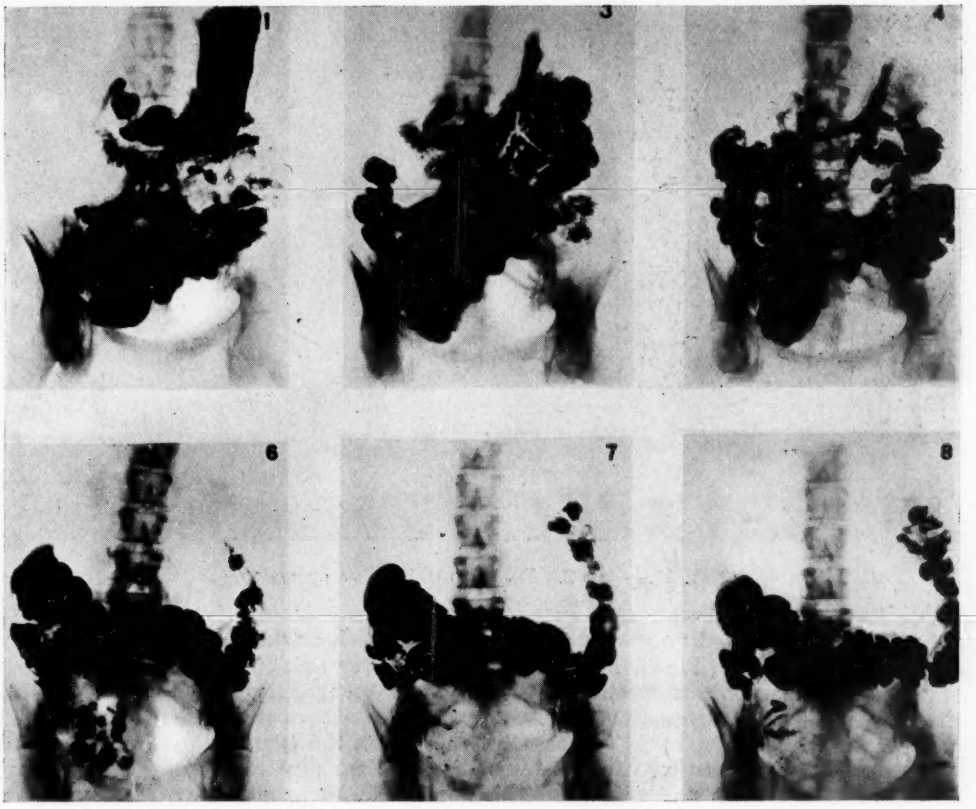

FIG. 12.—*Case* 1. Barium meal three weeks after operation.

work. She complained of a constant dull ache in the right flank and iliac fossa, increased by fatigue, and sometimes very severe. She always had an evening temperature.

On Examination.—The patient was admitted to New Lodge Clinic for investigation. While under observation her temperature rose every night to between 99° and 99·8°. The leucocyte count before admission showed 12,000 white corpuscles per c.mm., with 74 per cent polymorphonuclear cells: in the clinic three counts showed a less marked, but definite, leucocytosis, with a relative increase in polymorphonuclear cells. The red-cell count on admission was 3,700,000 cells per c.mm., and the hæmoglobin percentage 60. After daily injection of iron and arsenic for three weeks, the red count only rose to 4,080,000 cells per c.mm., and the hæmoglobin percentage to 66.

A fractional test-meal showed a marked deficiency in free hydrochloric acid.

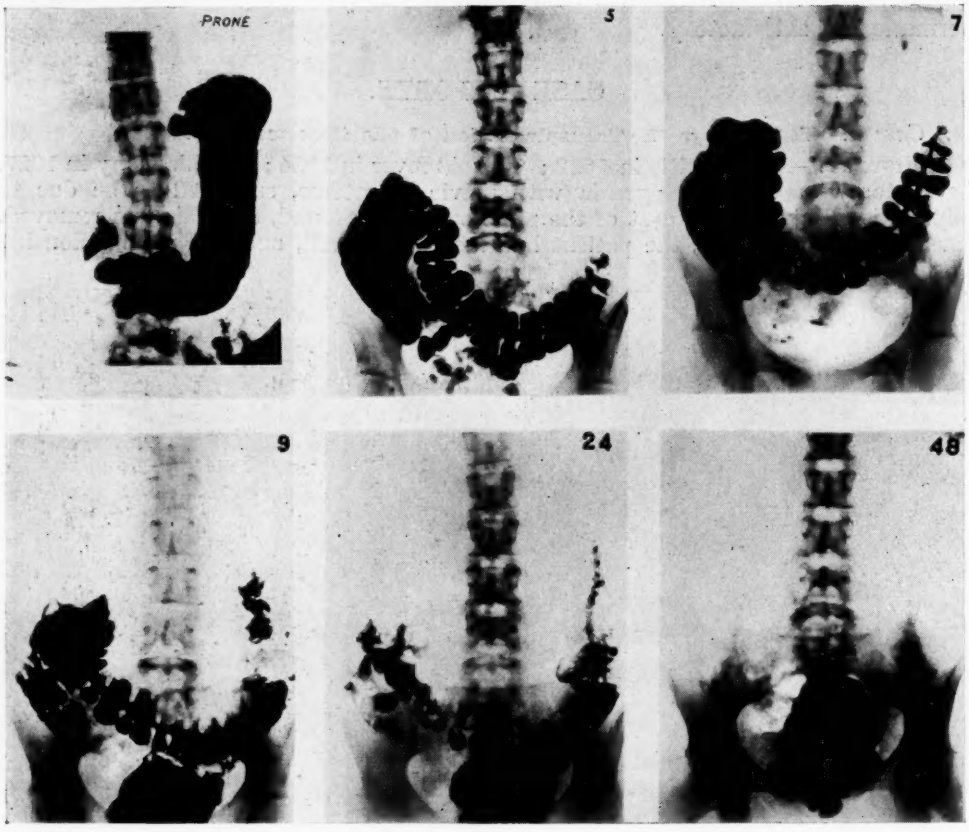

Fig. 13.—*Case* 2. Barium meal before operation.

A barium meal showed no abnormality in the stomach or duodenum; there was no abnormality in the outline of the large or small intestine, and no delay. By palpation under the fluorescent screen when the colon had been made visible, it appeared that the tenderness was confined to the cæcum and ascending colon; the cæcum, which just dipped into the true pelvis, was less tender than the ascending colon.

Repeated examination of the stools disclosed no abnormality. Cultures gave *Bacillus coli communis* and enterococci in normal numbers; no other organisms were found. A prolonged search failed to reveal amœbic cysts or tubercle bacilli. There was no excess of food residue, no excess of mucus or epithelial cells, no pus cells, and no blood, either obvious or occult.

A catheter specimen of urine showed no abnormality, and cultures remained sterile. Examination by a gynæcologist failed to reveal any cause in the pelvis to account for the pain or the pyrexia.

A diagnosis of chronic infection of the ascending colon was made. The patient was kept in bed on a non-residue diet, and her bowels regulated by ½ oz. of paraffin three times a day. A drachm of dilute hydrochloric acid was also given three times daily. In addition she was given *acidophilus* culture, charkaolin, bismuth salicylate, and belladonna. She was also given diathermy to the right side of the abdomen. No improvement resulted from this treatment.

OPERATION (May 30, 1930).—Spinocain, followed later by ether. Resection of ascending and one-third of transverse colon: transplantation of ileocæcal sphincter into transverse colon.

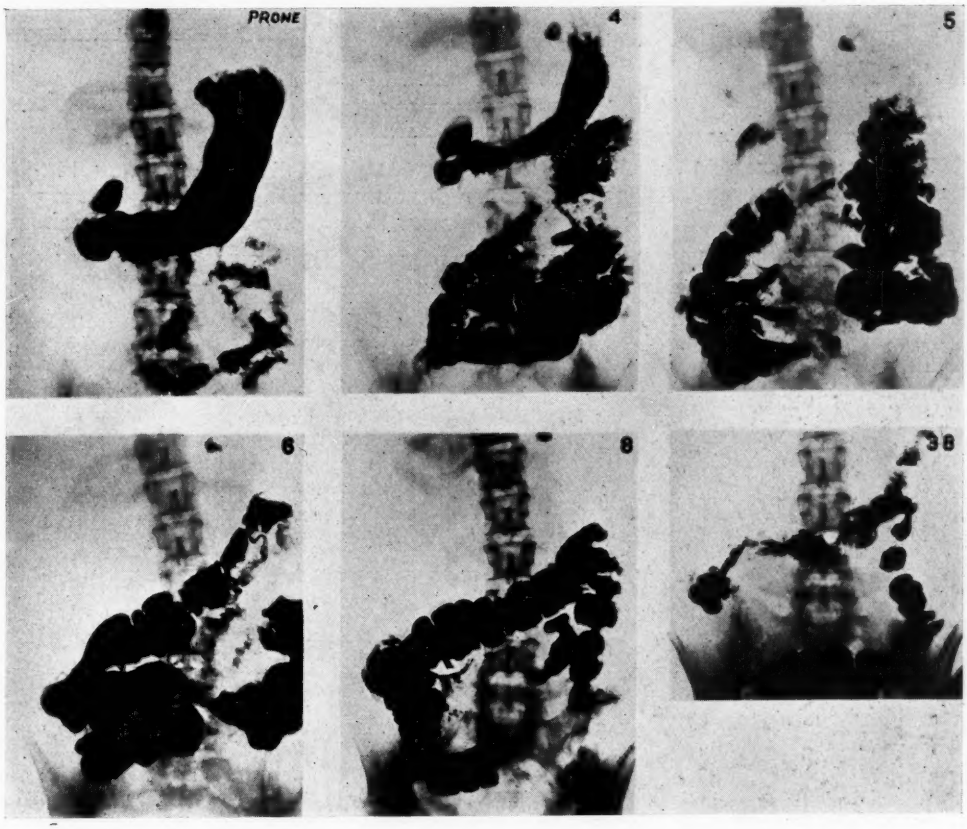

FIG. 14.—*Case* 2. Barium meal three weeks after operation.

REPORT ON SPECIMEN REMOVED (Dr. G. W. Rake).—Three piece of large intestine. The largest portion includes the whole circumference of the wall of the colon and measures 7 cm. in length. The longitudinal muscle bands are firmly contracted so that they are readily visible and palpable as raised white firm strands. The serous surface is thick, white, and opaque. There are several small adhesions, and one larger one which has been ligatured. The subserous adipose tissue is about normal in amount. In one place is a subserous hæmatoma the size of a cherry-stone, apparently resulting from the surgical procedure since it lies in close relation to a catgut suture. The muscular coat is not thickened and appears normal; the submucosa is somewhat thickened. The mucosa is free from all sign of inflammatory change or ulceration. The lymphoid follicles are normal in size and number. The mucosa shows a general dark-brown colour with irregular areas of a darker grey pigmentation; it is not thickened.

The two smaller portions have the same general appearance as the larger. Microscopical examination shows a very advanced degree of pigmentation in the mucosa. The pigment, which is in the form of coarser and finer, golden-brown, melanin-like granules, lies mostly within large mononuclear cells, although part is apparently free in the supporting stroma of the mucosa. It is most abundant just within the muscularis mucosæ, but extends up, in decreasing amount, between the glands to the surface. Epithelium and lymph follicles remain free from the pigment. The mucosa is otherwise normal. The submucosa is slightly thickened, with increase of fibrous tissue. The vessels show a slight degree of endarteritis. The muscle coats

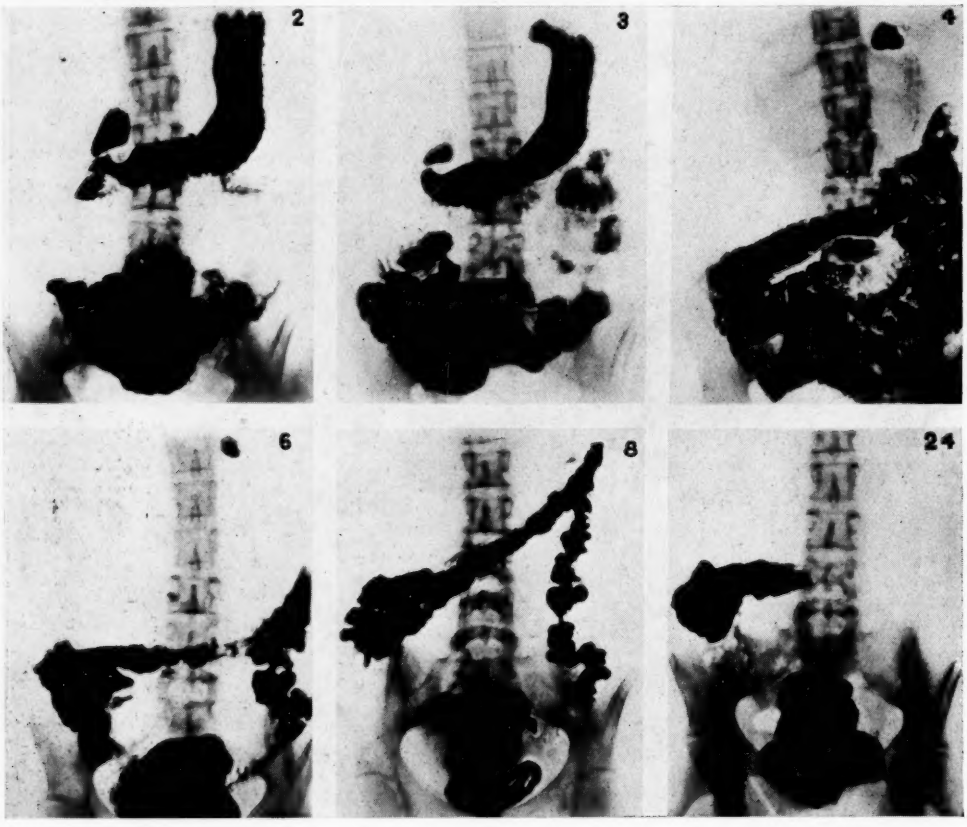

FIG. 15.—*Case* 2. Barium meal four months after operation. In all three series of this case a developmental pouch can be seen near the cardia.

appear normal; the plexus shows no abnormality. The subserosa is slightly thickened and fibrous. In one localized area in the muscle coat, and, to a lesser degree, in the submucosa, the capillaries are surrounded by a zone of polymorphoneutrophil and eosinophil leucocytes together with monocytes. This is obviously of very recent origin. Its causation is not clear.

The (so-called) melanosis of the colon is of a severe degree such as is of infrequent occurrence. As far as I know such a degree of change is almost invariably associated with severe constipation.

DIAGNOSIS.—Melanosis of colon.

SUBSEQUENT PROGRESS.—

Barium meal (three weeks after operation—*Fig.* 12).—The new cæcum lies in the right hypochondrium. Barium reaches the colon in four hours, and the

majority has left the ileum in six hours. A small residue remains in the last two inches of ileum at eight hours.

Six months after operation the patient is feeling very much better and leading an active life. Some anæmia persists and she has not yet returned to work. She has had no abdominal pain or pyrexia since the operation.

Case 2.—**Female, age 24. Transplantation of the ileocæcal sphincter.**

HISTORY.—Operation for gall-stones in 1923; gall-bladder and appendix removed. Second operation for adhesions in 1926. Laparotomy in 1927, right

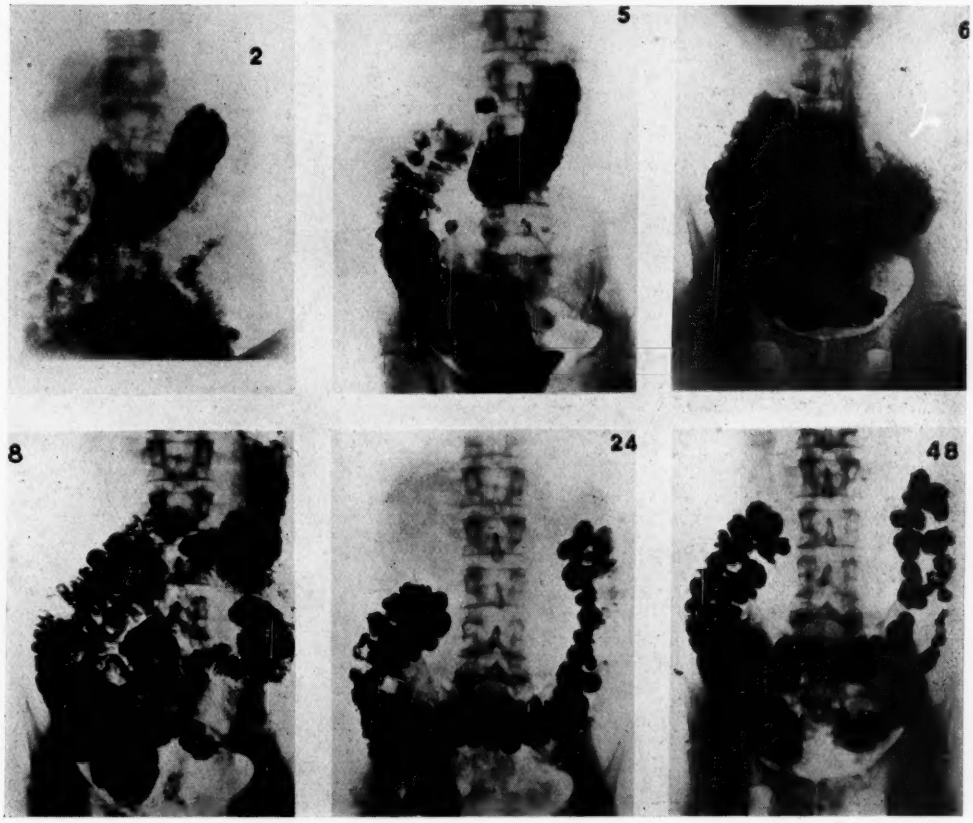

FIG. 16.—*Case* 3. Barium meal before operation.

ovary removed. The patient was admitted to New Lodge Clinic in 1928 for pain in the gall-bladder region; this cleared up under treatment with olive oil and tincture of hyoscyamus half an hour before meals. Shortly after this she developed a new pain low down on the right-hand side of the abdomen, over the site of the cæcum and ascending colon. The pain was constantly present, and bore no relation to the action of her bowels; it was not aggravated by meals or exercise, nor at the menstrual periods. The pain gradually got worse, and led to exhaustion, insomnia, and loss of weight. She was obliged to give up all games.

ON EXAMINATION.—She was readmitted to New Lodge Clinic in 1930. Marked tenderness was found over the cæcum and ascending colon, and over this area there was a good deal of muscular guarding.

A barium meal (*Fig.* 13) showed a diverticulum near the cardiac end of the stomach, causing no symptoms; there was no deformity of the ileum or cæcum,

and no stasis. When the parts were visualized it was shown that tenderness was most marked over the end of the ileum, and the cæcum; there were no shadows in the right iliac fossa suggestive of tuberculous glands.

The stools contained no occult blood and no tubercle bacilli. A catheter specimen of urine showed no abnormality. The red-cell and white-cell counts were normal, and the differential white-cell count showed no abnormality. A fractional test-meal showed hyperchlorhydria and rapid emptying.

The patient was kept in bed for three months on a non-residue diet, and her bowels regulated with liquid paraffin. The pain was not improved.

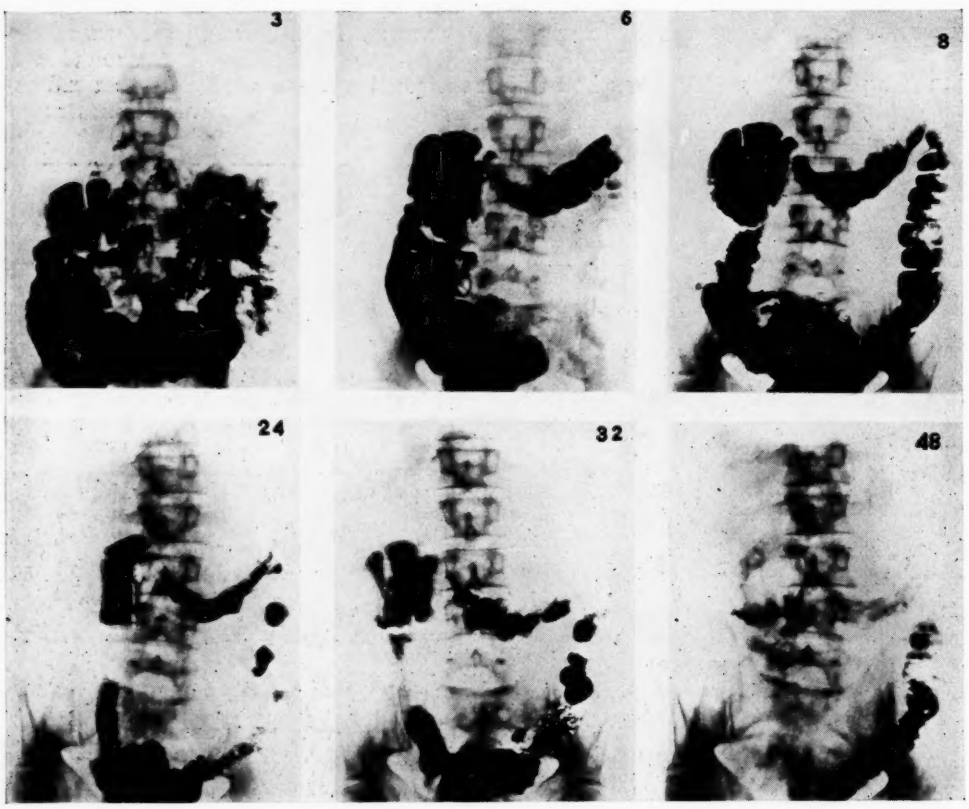

FIG. 17.—*Case* 3. Barium meal two and a half weeks after operation.

OPERATION (Sept. 16, 1930).—Laparotomy under ether anæsthesia. Very extensive adhesions were found, binding the cæcum and ascending colon to the parietal peritoneum of the right iliac fossa and anterior abdominal wall. The cæcum, ascending colon, and half of the transverse colon were removed, and the ileocæcal sphincter was transplanted laterally into the transverse colon 2 in. beyond its blind end.

SUBSEQUENT PROGRESS.—

Barium Meal (three weeks after operation—*Fig.* 14).—There is a certain amount of delay in the passage of the meal at all stages: some residue remains in the stomach at five hours, and a considerable amount occupies the lower coils of the ileum at eight hours. The new cæcum lies in the right flank at the level of the iliac crest: the transverse colon runs an almost straight course.

Barium Meal (four months after operation—*Fig.* 15).—The passage of the meal through the alimentary canal is normal. All the barium has left the small intestine in seven hours: a residue remains in the new cæcum at twenty-four hours.

Four months after operation the patient is feeling well, is leading an active life, and has put on 10 lb. in weight. There is a little tenderness on deep pressure in the right iliac fossa.

Case 3.—Male, age 39. Transplantation of cæcum.

History.—Complained of recurring attacks of severe abdominal pain, from which he had suffered for twenty years. He had these attacks every three or four months, and each attack was identical and lasted about ten hours. He was always constipated during an attack. Pain commenced with a dull ache in the gall-bladder

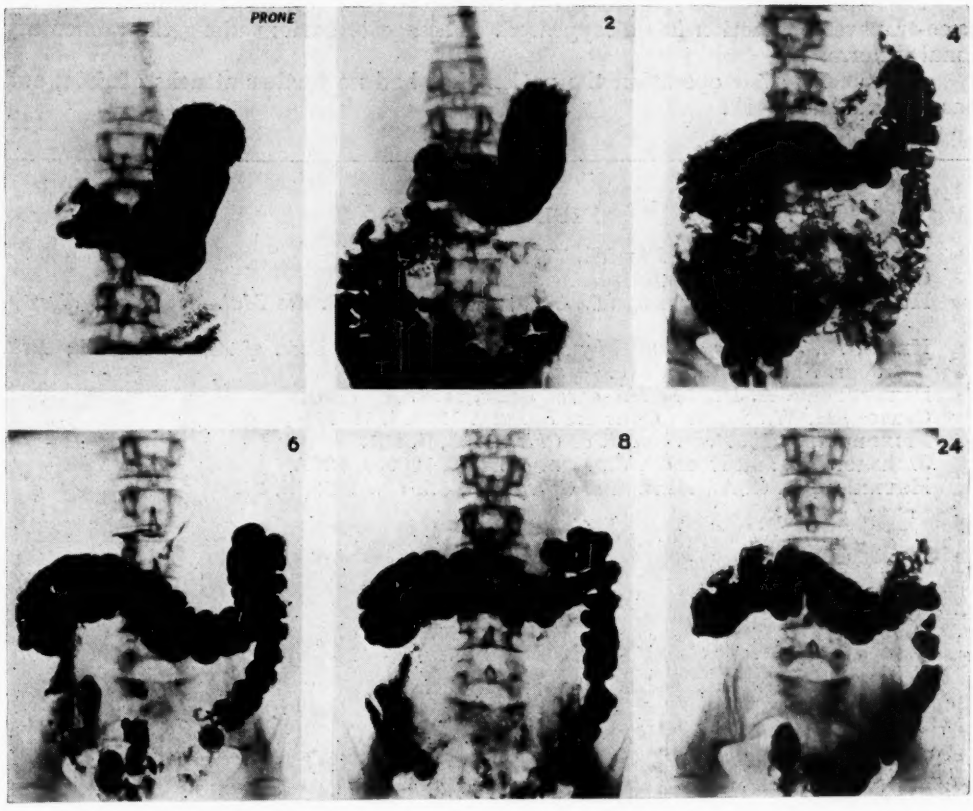

Fig. 18.—*Case* 3. Barium meal three months after operation.

region, which gradually got worse, till it was very severe. At the height of the pain he vomited, but without any relief. After an enema the bowels were opened, and the pain rapidly subsided, being nearly gone in three hours, but leaving a soreness behind for the whole of the next day. In 1925 laparotomy was performed and a peritoneal band running from the gall-bladder to the duodenum was divided; the appendix was also removed. After this operation he remained free from pain for more than a year, but since that time the attacks have recurred as before. Latterly he has passed blood per rectum during an attack.

On Examination.—No abnormality was discovered on physical examination, beyond the scar of the old laparotomy, and that of a previous herniotomy. A barium meal (*Fig.* 16) showed the stomach, duodenum, and small intestine to be normal. The transverse colon hangs down in a pronounced loop, its proximal third running vertically downwards parallel to the ascending colon, so that the hepatic flexure

is sharply angulated. The majority of the meal is still in the colon at forty-eight hours, the cæcum still being full. There is no evidence of localized obstruction.

OPERATION (June 16, 1930).—Resection of ascending, and half of transverse colon : anastomosis of cæcum to transverse colon.

SUBSEQUENT PROGRESS.—

Barium Meal (two and a half weeks after operation—*Fig.* 17).—The cæcum lies in the right hypochondrium and is markedly distended, being separated from the transverse colon by a narrowed segment. The transverse colon now lies almost horizontally. Barium remains in the lower ileum up to twenty-four hours, and in the cæcum up to thirty-two hours.

Barium Meal (three months after operation—*Fig.* 18).—The diaphragm at the cæco-transverse junction has disappeared. Passage of barium along the alimentary canal is normal.

Six months after operation the patient has had no further attacks of pain, and has returned to work.

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CHRONIC FOLLICULAR GASTRITIS: WITH A REPORT OF NINE CASES.*

By R. R. FITZGERALD, Montreal.

Although it has long been the custom to associate chronic ulcer of the stomach and duodenum with persisting indigestion accompanied by periodic pain, vomiting, hæmorrhage, retention, and changes in the chemical content of the gastric juice, it is now definitely established that these signs and symptoms may occur in the absence of ulcer. It has been proved that chronic gastritis (simple non-specific inflammatory disease of the stomach wall) can itself be responsible for the complete 'pyloric syndrome'.

The studies of Konjetzny, Nicolaysen, and others in the last few years have directed attention to one particular form of chronic inflammation of the stomach wall, under the name of 'chronic follicular gastritis'. It is the purpose of this communication to review certain aspects of the subject of gastritis, and to place on record nine clinical cases of the chronic follicular type of the disease. In this series chronic indigestion and hæmorrhages were the prominent clinical features. The cases were all treated by partial gastrectomy, and in the gross specimens no ulcers were found in the portions of stomach removed at operation. There is no reason to believe that ulcers may have been overlooked at the time of resection. In every case there was found in the stomach wall a characteristic inflammatory change exhibiting the gross and microscopic features of 'chronic follicular gastritis'.

HISTORICAL.

About a century ago gastritis began to be mentioned in medical writings. Broussais is credited with the first description of the lesion. It is quite possible, however, that in the literature of that period post-mortem autodigestion was at times mistaken for a true organic lesion, and 'gastritis' perhaps appears in the autopsy records more often than it should were such a possibility ruled out. Fixation of the stomach immediately after death by the intraperitoneal injection of formalin served to establish the comparative rarity of true gastritis, but the authority of tradition was so strong that the error persisted in medical writings. Towards the end of last century the cellular pathology of the body organs was rapidly developed and correlated with the clinical signs and symptoms of disease, but post-mortem autodigestion

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excluded the stomach from satisfactory and convenient study until radical surgical procedures provided fresh material which could be placed in fixatives in the operating-room immediately after removal from the body. Up to twenty years ago only a few of the pathologists could speak with authority on the histopathology of the stomach, but during the last decade the surgeons have provided abundant material for the detailed study of the gross and microscopic appearance of the organ in various diseased states.

One outstanding result of these studies has been to bring to the fore the subject of gastritis. The proposal to group the cases showing the chronic follicular lesions as a separate type of gastritis has followed the recognition of its characteristic histopathology, associated with a particular type of clinical history, and a fairly constant response to radical surgical treatment by partial gastrectomy.

Hunger pains, hyperchlorhydria, and delayed emptying of the stomach formed the 'pyloric syndrome' of Soupault (1901). This group of symptoms, through the work of Moynihan and others, came to be definitely associated with chronic ulcer of the pyloric region. If vomiting and hæmorrhage were also present, a diagnosis of ulcer on clinical grounds alone was quite justified. It was not known that the pyloric syndrome could be present without ulcer until Konjetzny and other writers on this subject published their observations during the last few years.

Excluding syphilis, tuberculosis, and plastic linitis, Ewald distinguished three forms of gastritis: (1) Simple; (2) Mucous, or myxorrhœa gastrica; (3) Atrophic. The pathological changes which accompanied these three forms were numerous and varied. In chronic simple and in mucous gastritis the stomach was described as being usually large, and the mucous membrane pale and covered with tenacious mucus. Ecchymosis, pigmentation, and hæmorrhagic erosions were seen, and the surface was at times mammilated (gastritis polyposa). Parenchymatous degeneration and interstitial inflammation were seen under the microscope, especially in the pyloric region. In the advanced atrophic type the mucous membrane exhibited complete atrophy (pangastritis), and the muscularis mucosæ was hypertrophied. A generalized hardening and shrinking of the whole stomach was known to occur in late cases (cirrhosis of the stomach). Perigastritis (gastritis adhesiva) with organized adhesions to the surrounding structures was a recognized accompaniment of gastritis.

This summary of the conventional conception of gastritis does not include the clinical feature of *bleeding from the stomach*, or the histological finding of *lymph follicles with germinal centres* in the mucous membrane. Hale White described twenty-nine cases of hæmorrhage from the stomach without lesion of the mucosa under the name of *gastrostaxis*, and many other surgeons have observed blood oozing from multiple points of the apparently intact gastric lining. An association with gastritis was not suspected, and no histological reports on the cases are available. Lymph follicles with germinal centres are infrequently mentioned in the earlier descriptions of gastritis except in the writings of Stoerk and in those of a few other accurate observers.

A few years ago, under the influence of Einhorn and other clinical workers, the term *gastritis* came to take on a functional meaning instead of a true organic significance. It has been, and still is, used by many to indicate a neurosis, or the result of a neurosis. To avoid confusion it is used in this paper to denote a structural change in the stomach wall with the characteristics of chronic inflammation.

In 1913 Heyrowsky made a histological study of fragments of gastric wall which he had removed from the stomach during the operation of gastroenterostomy performed for the relief of retention in ulcer or cancer cases. In none of the specimens was normal mucosa found. An inflammatory change was constantly present, as evidenced by interstitial exudation and hyperplastic changes in the epithelium. When the technique of partial gastrectomy was brought to its present satisfactory state, studies were made of the part of the stomach removed for cancer or ulcer. These specimens always exhibited a gastritis associated with the primary lesion.

The next step in the development of the subject was the suggestion made by several surgeons, of whom Finsterer was among the first, that a partial gastrectomy should be carried out in those cases where the clinical history pointed emphatically to the presence of organic disease, but where no ulcer or cancer could be found at operation. This proposal was brought forward because several surgeons had performed successful and beneficial partial gastrectomies in cases where an external inspection of the stomach suggested that a chronic ulcer existed but an intact mucous membrane was found in the specimen after removal.

Konjetzny, in 1924, gave a detailed description of eight cases in which resection had been carried out in the absence of ulcer. The patients had all suffered from some variety of indigestion. Hæmorrhage was a frequent complication. Study of the specimens revealed no ulcer, but definite gross and microscopic evidence of disease, and his work led to the recognition of *chronic follicular gastritis* (the name was suggested by Dobrovolski) as a clinical and pathological entity. Reports of other cases soon followed, and it is now established that the pyloric syndrome can be present without ulcer. Faber goes so far as to state that the pyloric syndrome can no longer be considered to be caused by ulcer, but by juxtapyloric gastritis. He believes that the symptoms and signs constituting this syndrome are only to be interpreted as evidence of disease in the pyloric region.

Definition.—By 'chronic follicular gastritis', then, is meant an inflammatory change in the stomach wall of long duration, with variable gross characteristics but constant microscopic appearances, and a clinical course marked by dyspepsia and often by hæmorrhage. The histological picture is that of inflammatory exudation into the mucosa. The exudate consists of eosinophils, plasma cells, and many lymphocytes, which are aggregated in places to form large lymph follicles with germinal centres. The lesion is probably related to the follicular form of duodenitis, but for the purpose of the present discussion is considered a separate lesion. The etiology of chronic follicular gastritis, and the relation between it and chronic gastric ulcer, are still unsolved problems.

CASE REPORTS.

The microscopic study of the sections from the following cases is based upon tissue which was fixed at the operation, or soon afterwards, embedded in paraffin, and the sections stained with differential stains.

Case 1.—Miss I. G., age 56, housekeeper. Admitted April 19, 1928, complaining of belching of gas.

History.—At the age of 15 she was first troubled with epigastric pain, accompanied by flatulence, coming on about two hours after meals. The attacks would last about a month at a time, and occurred about twice yearly. Ten years ago this indigestion ceased, and she was free from symptoms for five years. She then began to complain of eructation of gas, with no periodicity or relation to the taking of food. No hæmorrhage. Nutrition maintained.

On Examination.—Test-meal gave a small quantity of gastric content with low acidity (4·6 to 8), traces of blood, no free HCl. No blood in the stool. X-ray examination showed an interruption of peristaltic waves in the pyloric region, with a filling defect, and six-hour retention.

Clinical Diagnosis.—Carcinoma engrafted upon ulcer.

Operation (May 3—Dr. W. L. Barlow).—Stellate scarring noted on the anterior surface of the pylorus. There was also a slight hour-glass deformity due to thickening and shortening of the gastro-hepatic omentum. An enlarged lymph gland was noted in the gastrocolic omentum. There were adhesions in the lesser peritoneal sac between the stomach and pancreas. Partial gastrectomy (Polyá method).

Pathological Report (S–28–566).—The specimen consists of a portion of stomach 12 cm. in length and 4·5 cm. in width before opening. The inner surface shows no evidence of any ulceration.

Microscopic Appearances.—In many areas the mucous membrane is so intensely infiltrated with inflammatory cells (plasma, round cells, and eosinophils) with large germinal centres, that the gastric glands cannot be recognized. Dilated blood-vessels appear in large numbers in the mucosa and submucosa. There is extreme thickening of the muscularis mucosæ. The submucous area is entirely replaced by fibrous tissue. There are many large blood-vessels with thickened walls in the subserosa. Follicular gastritis.

Subsequent Course.—Twenty-nine months after operation (October, 1930), general health excellent; no indigestion. Works as a domestic servant.

Comment.—The X-ray findings pointed to organic disease, and deformity of the stomach was found at operation, but microscopic study of the specimen showed no ulcer or cancer. A typical picture of chronic follicular gastritis was found.

Case 2.—Mr. C. S., age 34, clerk. Admitted Dec. 18, 1921.

History.—Indigestion for six months: at first a feeling of fullness, then eructations of acid-tasting material. One week previously he felt a sudden heavy weakening pain in the epigastrium which usually came on one or two hours after food. Slightly nauseated. Sometimes the pain would appear about one hour before meals and be relieved by eating. On the day of admission there was a sudden stabbing pain in the upper abdomen, spreading to the back and right shoulder.

On Examination.—There was a general rigidity over the right upper quadrant of the abdomen.

First Operation (Dec. 18—Dr. A. Hutchison).—A perforation was seen on the anterior wall of the duodenum, with free escape of fluid; this was closed. Posterior gastro-enterostomy. The patient developed pleurisy with effusion six days after operation, but made a good recovery, and was discharged 'well' on Jan. 5, 1922.

Re-admission (Oct. 28, 1928).—Almost seven years after operation the patient consulted his physician on account of tarry stools. The following day the stool

contained dark blood, and he experienced a feeling of profound weakness. There was a severe secondary anæmia (Hb 55 per cent); a barium meal showed a functioning gastro-enterostomy, but no evidence of gastric ulcer, cancer, or marginal or jejunal ulcer.

SECOND OPERATION (Nov. 13—Dr. W. L. Barlow).—Numerous adhesions divided between transverse colon, omentum, and anterior abdominal wall, but no adhesions noted in the region of the duodenum. No external evidence of ulcer. A pouch-like projection on the lower surface of the first part of the duodenum indicated the site of the previous ulcer. Thickening of the pyloric ring was noted. The epiploic foramen was obstructed. Pylorectomy, with removal of the first part of the duodenum.

PATHOLOGICAL REPORT (S–28–1369).—The specimen consists of a portion of stomach and duodenum, with a few adhesions in the region of the pylorus. Pyloric ring thickened. There are about half a dozen areas of hæmorrhage in the mucosa of the stomach; but no ulceration, acute or old, can be made out.

Microscopic Appearances.—Lymph follicles with germinal centres are present in the mucosa. The gland tubules are separated by round cells, plasma cells, and eosinophils. At the pylorus there is a diffuse fibrosis of the deepest layers of the mucous membrane, and there are very few gland tubules or acini to be seen. There is a pronounced follicular duodenitis.

DIAGNOSIS.—Chronic follicular gastritis and duodenitis.

SUBSEQUENT COURSE.—Twenty-three months after operation (October, 1930), excellent general health, no indigestion, no hæmorrhages. Cured.

Comment.—The interest in this case centres around the sudden hæmorrhages without indigestion which came on just before the second admission to hospital.

Case 3.—Mr. R. B., age 37. Admitted Oct. 7, 1928.

HISTORY.—There was a history of a gnawing burning epigastric pain of ten years' duration. At times a dull aching pain was felt in the chest. The pains originally came on at rare intervals, but they had been steadily increasing in frequency and duration. Vomiting frequently after solid food; no bleeding. Obstinate constipation. Pains were relieved by taking water or soda, and aggravated by food. Loss of 40 lb. weight in a few months.

ON EXAMINATION.—First barium series, negative. Second barium series showed evidence of a duodenal ulcer with 50 per cent retention at six hours. Test-meal, 75 c.c.; total acidity 78 per cent, free HCl 56 per cent. No occult blood in gastric contents or stool.

Sippy régime begun. Relief of all pain in twenty-four hours; vomiting stopped; hæmoglobin rose from 78 to 105 per cent. Discharged Nov. 7, feeling well except for a sensation of weight in the epigastrium.

RE-ADMISSION (Nov. 17).—The patient complained of a return of vomiting and a distressing feeling of weight in the region of the heart.

OPERATION (Nov. 24—Dr. C. K. P. Henry).—Removal of atrophic kinked appendix. Stomach and duodenum normal on inspection. In view of history and X-ray findings, partial gastrectomy (Polyá).

PATHOLOGICAL REPORT (S–28–1416).—The specimen consists of a portion of the pyloric end of the stomach. The mucosa is covered with tenacious mucus. It is pale, with a few small patches of petechial hæmorrhage. The pyloric ring is not thickened; there is no ulcer.

Microscopic Appearances.—Mucosa shows abundant follicles which are placed close to the muscularis mucosæ. Very little infiltration of the mucosa. No thickening of the muscularis mucosæ. In the muscular coat there are many collections of inflammatory cells (polymorphs) arranged about small blood-vessels. Collections of plasma cells, round cells, and eosinophils are also found in the subserous layer.

DIAGNOSIS.—Chronic follicular gastritis.

SUBSEQUENT COURSE.—Twenty-three months after operation (October, 1930), no pain or vomiting; gain in weight 37 lb. Working as upholsterer. Very slight epigastric discomfort after indiscretions in diet or heavy exertion. Cured.

Comment.—A thorough trial of conservative treatment failed to control the symptoms. The microscopic examination of the stomach revealed surprisingly little change in the mucosa, when the duration and the severity of the symptoms were considered. Collections of inflammatory cells were found in the muscularis and subserosa.

Case 4.—Mr. J. P., age 40, labourer. Admitted Nov. 19, 1928, complaining of abdominal pain and vomiting.

History.—Nine months before admission he suffered an attack of abdominal pain with vomiting lasting one week. Since that time had complained of epigastric pain, anorexia, and vomiting. There were alternate periods of diarrhœa and constipation. The pain was unaffected by the taking of food. Considerable loss of weight.

On Examination.—Slight splinting of the abdominal muscles in the epigastrium and right upper quadrant was revealed, and there was definite tenderness on pressure over the upper part of the right rectus muscle. X-ray negative.

Operation (Oct. 26—Dr. C. K. P. Henry).—The duodenum was covered by strands of fine vascularized adhesions. In view of the clinical history a partial gastrectomy (Polyá) was performed. Patient discharged nineteen days later; no complaints.

Pathological Report (S–29–1425).—The mucous membrane shows infiltration with wandering cells (plasma cells, round cells, eosinophils), intense in some areas. There are many follicles with germinal centres occupying the intermediate and deepest layers of the mucosa. The muscularis mucosæ is much thickened, and, in places, split into layers. There is a mild follicular duodenitis.

Diagnosis.—Chronic follicular gastritis.

Subsequent Course.—Working as a labourer (October, 1930); very rough food; very good health; free from indigestion. Cured.

Comment.—The clinical history of previous acute illness suggested penetrating ulcer. At operation the stomach and duodenum appeared normal. A resection was carried out on account of the definite clinical history.

Case 5.—Mrs. A. M., age 47. Admitted Feb. 4, 1929.

History.—The patient complained of recurring attacks of abdominal pain, accompanied by vomiting of large quantities of watery brown material. The last attack was particularly severe and occurred on the day of admission.

On Examination.—The abdominal muscles were splinted, and there was an acutely tender area in the epigastrium, just to the left of the mid-line. Pulse 80, temperature 99·4°.

Diagnosis.—Penetrating peptic ulcer.

Operation (Feb. 4—Dr. A. Stewart).—Gastro-hepatic omentum adherent to the anterior abdominal wall. This was separated and it was found that the same portion of omentum was firmly adherent to the lesser curvature of the stomach about midway between the pylorus and cardia. The gastric wall was thickened and œdematous at this point. Diagnosis: perforating gastric ulcer sealed by omentum. Partial gastrectomy, 'Balfour' method, with removal of the supposed ulcer.

Pathological Report (S–29–134).—No ulcer seen.

Microscopic Appearances.—The mucous membrane is packed with inflammatory cells (round cells, plasma cells, eosinophils). There are many follicles with germinal centres. The follicles are very large, and reach almost to the surface. There are many crypts or defects on the mucous surface leading down to the follicles or their neighbourhood. There is an irregular thickening of the muscularis mucosæ. In the subserosa there is œdema and infiltration with inflammatory exudate. One cyst seen.

Diagnosis.—Follicular gastritis.

Subsequent Course.—A period of severe vomiting followed the operation, requiring gastric lavage and intravenous salines. The patient left the hospital on

the twenty-ninth day after operation in very good condition. She is entirely free from digestive complaints eighteen months after operation.

Comment.—In this case the acute onset and the severity of the symptoms suggested penetrating ulcer with peritonitis. No ulcer could be found in the specimen.

Case 6.—Mr. H. C., age 32, mechanic. Admitted June 20, 1928.

History.—Had suffered for four years with dull aching abdominal pain which was continuously present day and night, but was always worse when he was hungry. Food relieved the pain for about half an hour. He brought up a mouthful of vomitus ten or twelve times a day. Blood in the stool at times. Eructations of gas. Lost 23 lb. in weight in five years. Sippy and Lenhartz diets tried without relief. Mother has stomach trouble. Father, age 65, has had two operations for a stomach ulcer. One sister is receiving treatment for gastric ulcer.

On Examination.—A sharply localized point of tenderness was revealed in the epigastrium. Test-meal gave: total acidity 80, free HCl 60. Blood was detected by chemical tests of the stool. Barium series revealed a constantly deformed cap, hyperperistalsis of the stomach, and rapid advance of the meal.

Clinical Diagnosis.—Duodenal ulcer.

First Operation (June 25—Dr. C. K. P. Henry).—Adhesions found between the great omentum, cæcum, transverse colon, hepatic flexure, liver, gall-bladder, and anterior abdominal wall. Extensive periduodenal adhesions, and a suggestion of an ulcer on the anterior wall. Pyloric ring crushed, and ligated with silk. Posterior gastro-enterostomy. There was a slight wound infection and vomiting at intervals after the operation. Discharged on Aug. 6.

Re-admission (March 25, 1929).—Complained of abdominal pain which came on one or two hours after meals, relieved by the next meal. Vomiting came on fifteen to twenty minutes after each meal. The pain was often nocturnal. Test-meal gave a volume of 39 c.c., total acidity 53·5, free HCl 39·5.

Second Operation (April 2—Dr. C. K. P. Henry).—Adhesions previously encountered had been partly absorbed. The pyloric part of the stomach was hard and tubular. Removal of the pyloric portion of the stomach and first part of the duodenum without interfering with the gastro-enterostomy.

Pathological Report (S–29–382).—The specimen consists of the pyloric part of a stomach and first part of duodenum. The mucosa shows no ulceration.

Microscopic Appearances.—In the superficial layers of the mucous membrane the glands are separated by a slight but definite amount of cellular exudate, made up chiefly of plasma cells, but containing a few round cells and eosinophils. In the deeper layers there are large follicles with germinal centres. The pyloric musculature shows a large irregular scar (ligature).

Diagnosis.—Chronic follicular gastritis.

Subsequent Course.—Nineteen months after operation (October, 1930) he was feeling quite well and free from indigestion.

Comment.—Pyloric occlusion and gastro-enterostomy failed to control the symptoms, and the patient returned in nine months for the relief of periodic pain and vomiting.

Case 7.—Mr. E. R., age 52, machinist. Admitted Feb. 21, 1927.

History.—Attacks of aching pain in the epigastrium for fifteen years. At first the pain came on two or three hours after eating, and was relieved by food. For the past few years the pain had no relation to the taking of food. The attacks came on four or five times yearly, with long periods of relief. Attacks were always preceded by diarrhœa. No bleeding.

On Examination.—Test-meal: 25 c.c., total acidity 15, free HCl 1. Occult blood present. Discharged on medical treatment.

RE-ADMISSION (Jan. 20, 1929).—Complaining of abdominal pain, loss of weight, weakness, anorexia. Slight anæmia (Hb 72 per cent). Occult blood detected in stool. Test-meal gave 59 c.c., total acidity 12, no free HCl. Occult blood present in stomach contents. X-ray showed a small ulcer of the duodenum. He was given the Sippy régime, and left the hospital in twenty-eight days without pain.

SECOND RE-ADMISSION (Aug. 10).—Felt well until a month ago when there was a return of the periodic pain, which was always relieved by food. Epigastric tenderness.

OPERATION (Aug. 13—Dr. C. K. P. Henry).—Stomach and duodenum normal except for a small thickening of the duodenal wall. Partial gastrectomy.

PATHOLOGICAL REPORT (S–29–1190).—The specimen consists of a portion of a stomach measuring 7 by 4 cm. Mucosa covered with a viscid secretion. There is no ulcer.

Microscopic Appearances.—The mucous membrane is closely packed with large follicles with germinal centres, and the gastric glands are separated from each other by round cells, plasma cells, and eosinophils. The muscularis mucosæ is irregularly thickened and fibrosed. The submucous layer shows diffuse scarring and dilated blood-vessels. There is also a follicular duodenitis.

DIAGNOSIS.—Chronic follicular gastritis and duodenitis.

SUBSEQUENT COURSE.—The operation was followed by intractable vomiting with forty-eight hour retention. Abdomen opened, adhesions divided. Discharged Sept. 21, feeling very well. Three months after the last operation there was still gastric retention (10 per cent in six hours). Classified as improved.

Comment.— Prolonged conservative treatment failed to control the disease. Considerable loss of blood and anæmia were important associated conditions.

Case 8.—Mr. C. B., age 32, chauffeur. Admitted Nov. 5, 1929.

HISTORY.—Complained of attacks of colicky pain and eructations of gas, lasting from a few minutes to three hours. No relation to the taking of food. Began eleven years ago, following an attack of influenza. Periods of relief of several weeks' duration. The pain was felt along the right costal margin. Appetite good. Tarry stools. Temporary relief with soda treatment.

ON EXAMINATION.—Total acidity 84, free HCl 41. Gall-stones revealed by cholecystogram. Diminished sugar tolerance. Barium series showed evidence of a duodenal ulcer.

OPERATION (Nov. 21—Dr. E. M. Eberts).—Cholecystectomy for gall-stones. Periduodenal adhesions were noted. The external appearance of the duodenum strongly suggested the presence of an ulcer. Partial gastrectomy (Polyá) with removal of the first part of the duodenum.

PATHOLOGICAL REPORT (S–29–1600).—The specimen consists of the pyloric portion of the stomach and first part of the duodenum. The mucous membrane is intact. The stomach wall is somewhat œdematous. At the commencement of the duodenum there is a thickening of the wall about 1·5 cm. in length.

Microscopic Appearances.—The gastric mucosa is hyperæmic, and there is an interstitial exudate composed of plasma cells, lymphocytes, eosinophils, and in places a few polymorphs. There is an increase in the lymph follicles, which show large germinal centres. The muscularis mucosæ is thickened and infiltrated with inflammatory cells. In the muscular coat there are foci of inflammatory exudate. The duodenum shows a large collection of pancreatic acini scattered in the submucosa, muscularis, and subserosa. There is fibrosis of the serous coat overlying this area. There are several ducts in the pancreatic tissue. A small collection of Brunner's glands was also noted in the subserosa. A mass of smooth muscle was present in the mucosa near the pancreatic tissue.

DIAGNOSIS.—Chronic follicular gastritis, with misplaced pancreatic tissue in the duodenal wall. Patient discharged in twenty days in excellent general health.

RE-ADMISSION (May 9, 1930).—Complained of having had two attacks of crampy pains beginning at the level of the lower end of the abdominal incision, lasting two or three hours. X-ray showed only a normally functioning partial gastrectomy. Conservative treatment (abdominal massage) recommended.

DIAGNOSIS.—Intestinal obstruction from post-operative adhesions. Classified as improved.

Comment.—Of interest here is the simultaneous existence of gall-stones and gastritis, together with a pancreatic rest in the duodenum. Among the cases of gastritis treated by resection reported in the paper by Wanke there are two in which post-operative attacks of partial intestinal obstruction were noted. As in the present case, the symptoms were attributed to 'adhesions'.

Case 9.—Mrs. C. H., age 54. Admitted April 6, 1930.

HISTORY.—Complained of indigestion and pains in the region of the stomach. In spite of great care in the matter of diet she had suffered from attacks of indigestion all her life. The attacks would consist of abdominal distress and eructations of gas. They would last for about one week, and would then disappear completely for a few months. She had been as long as a whole year without pain. During the attacks the pain had a distinct relationship to the taking of food. Bed, rest, and Sippy régime relieved the pain for about a year.

ON EXAMINATION.—X-ray showed a deformity of the first part of the duodenum.

OPERATION (April 11—Dr. A. T. Bazin).—Dense scarring of the anterior surface of the duodenum. Dilatation of the whole duodenum. Partial gastrectomy (Polyá). Cholecystectomy.

PATHOLOGICAL REPORT (S–30–452).—The specimen consists of pyloric portion of stomach, pyloric ring, and a fragment of the duodenum. There is considerable thickening in the pyloric region. There is a broad depression in the gastric mucosa, 2 by 2·5 cm., near the pylorus.

Microscopic Appearances.—There is no evidence of chronic ulcer. There are many lymph follicles with germinal centres crowded in the mucosa. Irregular thickening of the muscularis mucosæ. Scars in the mucous membrane. Glands are separated by round cells, eosinophils, plasma cells, and blood capillaries. Increase in the number of capillaries in the submucous layer. The muscularis is thicker than usual. There are many distended lymphatics filled with homogeneous material in the submucosa.

SUBSEQUENT COURSE.—Six months after operation, great improvement in general health; no indigestion; no pain; no vomiting. Cured.

Comment.—The long duration of the disability and the slight impairment of the general health are prominent features of the case.

CLINICAL AND PATHOLOGICAL SUMMARY OF CASES.

In the nine cases of this series the average age at operation was 43·3 years, and there were six males and three females. A family history of gastric disease was given by one patient (*Case* 6). The average duration of the symptoms was 15·8 years; the longest case had lasted 44 years, the shortest 9 months. The onset was gradual in every case excepting *Case* 5, where it was so sudden as to suggest acute perforation. Indigestion was present in all except *Case* 2, in which copious hæmorrhage was the only sign. Actual pain was noted in seven of the nine case histories; vomiting in four. The periodicity of the symptoms was a feature of several of the cases, and in

Table I.—Showing Principal Features of

Case No.	Age and Sex	Duration of Symptoms	Indigestion	Pain	Vomiting	Hæma-temesis	Occult Blood in Stomach	Melæna	Occult Blood in Stool	Acidity
		Years								
1	56 F.	41	+	0	0	0	+	0	0	4/0
2*	39 M.	7½	0	0	0	0	0	+	+	
3	37 M.	10	+	+	+	0	0	0	0	78/56
4	40 M.	¾	+	+	+	0	0	0	0	
5	47 F.	6	+	+	+	0				
6*	33 M.	5	+	+	+	0	0	0	+	53/30
7	52 M.	17	+	+	0	0	+	0	+	12/0
8	32 M.	11	+	+	0	0	0	+	0	84/41
9	54 F.	44	+	+	0	0	0	0	0	

* This patient had previously

some instances the seasonal and daily variations in relation to the taking of food were exactly those found in chronic peptic ulcer.

Loss of blood is the most arresting clinical feature. It is recorded in five of the nine case reports. Melæna, occult blood in the gastric contents, and occult blood in the stool have all been found. Hæmatemesis was not observed. The amount of blood lost by one of the patients was alarming (*Case* 2). One patient (*Case* 8) reported having had tarry stools on several occasions. Konjetzny has reported a fatal case of hæmatemesis in which gastritis (no ulcer) was found at autopsy. In this connection it is interesting

NINE CASES OF CHRONIC FOLLICULAR GASTRITIS.

X-RAY FINDINGS	OPERATIVE FINDINGS	OPERATION	PATHOLOGICAL REPORT	INTERVAL SINCE OPERATION	END-RESULT
				Months	
Interrupted peristalsis at pylorus Six-hour retention	Two scars. Hour-glass deformity Enlarged glands Adhesions	Partial gastrectomy (Polyá)	Follicular gastritis	29	Cured October, 1930
Functioning gastro-enterostomy	Thickened pylorus Pouch in duodenal wall	Pylorectomy	Chronic gastritis and duodenitis	23	Cured October, 1930
(1) Negative (2) Duodenal ulcer with 50 per cent retention at six hours	Normal stomach and duodenum	Partial gastrectomy (Polyá)	Chronic gastritis	23	Cured October, 1930
Negative	Vascularized periduodenal adhesions	Partial gastrectomy (Polyá)	Chronic follicular gastritis	22	Cured September, 1930
	Perigastric adhesions: local thickening and œdema of stomach wall	Partial gastrectomy (Balfour)	Chronic gastritis	18	Cured August, 1930
	Thickened antrum	Pylorectomy	Chronic follicular gastritis	19	Cured October, 1930
Negative	Area of thickening in duodenal wall	Partial gastrectomy (Polyá)	Chronic follicular gastritis	14	Retention 10 per cent Improved
Gall-stones Duodenal ulcer	Periduodenal adhesions Local thickening of duodenal wall	Partial gastrectomy (Polyá) Cholecystectomy	Chronic follicular gastritis Pancreatic tissue in duodenal wall Gall-stones	6	Re-admitted for pain Diagnosis, adhesions Improved
Duodenal ulcer Duodenal diverticulum	Scarring and dilatation of duodenum	Partial gastrectomy (Polyá) Cholecystectomy	Chronic follicular gastritis Chronic cholecystitis	6	Cured October, 1930

undergone a posterior gastro-enterostomy.

to note that hæmorrhage from the urinary tract is common in granular pyelitis, where microscopic study of the diseased pelvic lining shows follicular structures (Paschkis) comparable to those of the stomach mucosa in gastritis.

Excessive secretion of mucus (myxorrhœa gastrica) was not noted in this series. It is, however, well recognized as an accompaniment of chronic gastritis, and the work of Kuttner has drawn attention to the existence of two forms, one intermittent, the other continuous.

Chemical examination of the gastric content after test-breakfast showed normal values in some cases, hyperacidity, hypo-acidity, or anacidity in others.

According to Zweig, the amount of diminution in acidity can be taken as an index of the degree of destruction of gastric mucosa. Hyperacidity is said by him to be present in the early stages, normal values in advanced stages, anacidity in the final stage.

The *X-ray findings* have been inconstant. In three cases (*Cases* 3, 4, and 7) negative results were reported. In four (*Cases* 1, 3, 8, and 9) a diagnosis of ulcer was made. *Case* 3 is interesting. The first barium series was negative. A second series, taken a few days later, showed radiological evidence of duodenal ulcer. At operation only follicular gastritis was found. Most of the writers on the subject report diminished gastric mobility. H. H. Berg found characteristic X-ray pictures in gastritis after the administration of a few cubic centimetres of barium mixture by mouth. A great increase in the number and size of the mucous folds was noted. G. Schwarz mentions multiple indentations of the greater curvature, and these indentations have been noted by other writers. It is stated by Zweig that an increased sensitiveness to pressure (Druckempfindlichkeit) over the stomach outline, with multiple indentations along the greater curvature, and an increase in the number and depth of the folds, indicate the presence of gastritis.

Gastroscopy has proved disappointing as an aid to diagnosis. Although the technique is not difficult, the whole of the interior of the stomach cannot be clearly seen, and—what is more important—it is extremely difficult to interpret the findings correctly. As an example, Zweig writes of: (1) Petechial or streaky reddening; (2) Excessive mucus (catarrhus mucosus); (3) Gastritis hypertrophicans; (4) Multiple bleeding points and small erosions (gastritis hæmorrhagica and granulosa); (5) Gastritis atrophicans. Although it is possible to observe all of these changes through the gastroscope, their interpretation is difficult. B. Lubarsch has described changes in the stomach in purely physiological states that appear exactly like those found in gastritis. Schüller finds that by gastroscopy one can confirm the presence of gastric cancer in every case, but although he examined many cases of proved gastric ulcer, he was able to see the lesion itself only once. In gastritis cases he was often able to observe definite changes, but found difficulty in interpreting them, and in differentiating them from the physiological changes described by Lubarsch.

Table I (pp. 34, 35) shows the principal features of the nine cases of chronic follicular gastritis reported above.

PATHOLOGICAL ANATOMY.

Gross Appearances.—The aboral part of the stomach is usually the only portion of the organ affected. The greatest changes are found in the pylorus and pyloric antrum. The fundus is nearly always normal. Only in the advanced atrophic form of pangastritis with achylia gastrica (Faber) is the whole stomach uniformly diseased. The distribution of the lesion is not uniform, but decidedly uneven and patchy. Normal areas may be encountered interposed among portions of diseased gastric wall. Again, no two of the abnormal areas will be identical in a given case. It is indeed possible to find in different parts of one specimen, atrophy, excessive regeneration,

superficial ulcerations of the mucosa, polypoid overgrowth, and hæmorrhagic erosions.

External inspection may reveal a blush or reddening of the serous surface. Schoemaker specially emphasizes the importance of this sign, but it may be absent in true cases of gastritis. Perigastric adhesions may unite the stomach to the great omentum, the gastro-hepatic omentum, the liver, gall-bladder, colon, or anterior abdominal wall. In one recognized type the whole stomach appears to be embedded in organized adhesions (perigastritis adhesiva). The most constant gross finding is a thickening and a palpable hardening or stiffening of the stomach wall in the pyloric region. The antrum feels hard and tubular. Enlarged, soft, reddened lymph-glands are noted above and below the pylorus in about half the cases. Small diverticula are seen at times, always at the pylorus. These are generally interpreted as being the sequelæ of healed ulcers, but this is only an assumption, and one which is not always justifiable.

On opening the stomach there is seen a thick layer of slimy mucus adhering to the inner surface. No peptic ulcer can be found. Minute superficial hæmorrhagic erosions may or may not be present. When they are found they usually occupy the apices of slightly rounded projections, or they may appear as tiny slits between the folds of mucous membrane. They consist of small openings blocked by blood-clot. Superficial irregularly-shaped patches of partial destruction of the mucous membrane over circumscribed areas are often to be seen, especially in the pylorus and antrum. These areas are slightly pale, and are referred to in the literature as 'worm-eaten patches'. Active regeneration, hyperplasia, or compensatory overgrowth of the mucous membrane may lead to a papilliferous state of the mucous membrane. This process may be extremely advanced and may produce an 'état mammeloné' or gastritis polyposa. In the pyloric region this overgrowth of the mucous membrane may, by its bulk alone, become an important factor in the production of pyloric stenosis and retention. In two cases of this type Konjetzny found early cancer.

Microscopical Appearances (*Figs.* 19–23).—In mild cases the mucous membrane is the only layer of the gastric wall to show inflammatory change. In the advanced stages all the layers may be altered. The patchy distribution of the lesion is obvious in the histological sections. Neighbouring fields may show normal mucosa, chronic gastritis, atrophy, excessive regeneration, erosion, or mucosal ulcerations; and different parts of the same specimen will show great differences in the depth to which the disease has penetrated the stomach wall. In general, the changes are most marked at the pylorus and antrum. Seldom is the fundus involved.

The most striking, constant, and characteristic histopathological changes are to be seen in the mucous membrane. Here the tubules of the gastric glands are not in immediate contact with each other, but are separated by masses of inflammatory cells, small round cells, plasma cells, eosinophils, and connective-tissue cells. The small round cells are frequently aggregated to form follicles with large germinal centres. These lymphoid follicles are the outstanding characteristic of the lesion, and are responsible for the name 'chronic follicular gastritis'. The follicles vary in size, but are usually large

enough to occupy about half the thickness of the mucosa. Typically, they are circular or oval on section, but may be triangular or irregular in shape. The central portion, or germinal centre, is made up of rather poorly staining and fairly homogeneous cells of irregular shape, but all tending to be round or oval. About the germinal centre there is a collar of ordinary small, round or lymphoid cells.

The eosinophils of the interfoveolar inflammatory cell masses are often very abundant. Accompanying the interfoveolar cellular exudates the

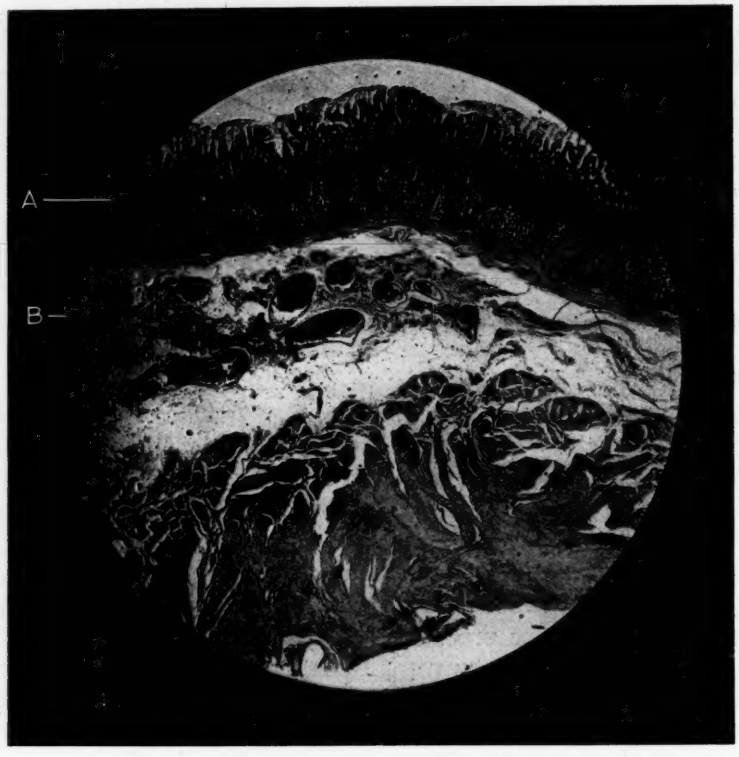

FIG. 19.—Chronic follicular gastritis, early stage. **A**, A row of lymph follicles with germinal centres embedded in the mucous membrane, and resting on the muscularis mucosæ, which is slightly thickened; **B**, Dilatation of the blood-vessels of the submucosa.

epithelial cells lining the gastric glands and their ducts show various changes. The surface epithelium, lining the actual gastric lumen, shows the most frequent alterations. These cells are normally mucus-secreting, and in sections from cases of follicular gastritis they are nearly always very active, and are filled with large masses of mucus. More advanced stages show atrophy and regressive changes, and, at times, hypertrophy or actual hyperplasia. The epithelium lining the mouths of the gastric glands is also mucus-secreting, and shows changes similar to those on the free surface.

In the normal human stomach the epithelial cells lining the elongated ducts of the gastric glands and the acini in the deepest layer of the mucosa are the true gastric secreting cells. They produce the gastric juice, and vary

in appearance in different parts of the stomach. Those in the pyloric region are simple cuboidal cells with opaque cytoplasm, and are believed to secrete a mucous material only. These pyloric gland cells are found at the pylorus itself, and in the pyloric canal and antrum. A few scattered lymphoid follicles with germinal centres are normally present in the mucosa of the antrum. Above the antrum the typical pyloric cells disappear rather suddenly, and in the body of the stomach the glands are lined by epithelial cells of three different types—namely, chief or oxyntic cells, parietal cells, and intermediary

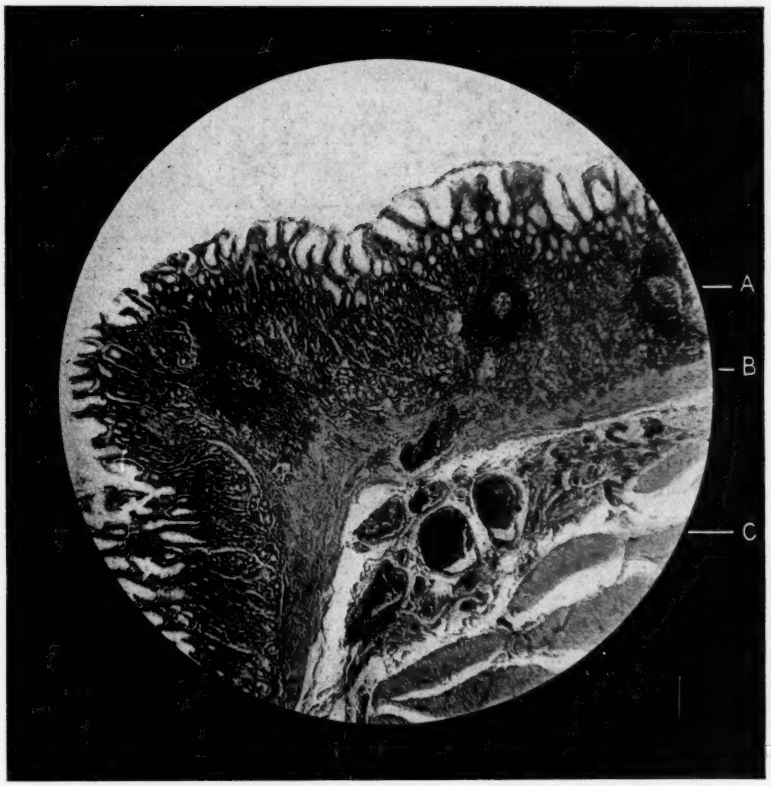

FIG. 20.—Chronic follicular gastritis. A, Three follicles with germinal centres; B, Thickening of the muscularis mucosæ; C, Dilatation of the submucous blood-vessels.

cells, the latter closely resembling the pyloric cells described above. These three varieties of cells are found throughout the greater part of the fundus and cardia, but about the cardiac orifice itself there is a narrow band of mucosa whose lining cells are morphologically similar to those of the pyloric region—that is, smaller cuboidal cells with cloudy cytoplasm (the cardiac cells).

The above description is that of the normal human stomach. In chronic follicular gastritis these specific secreting cells often show surprisingly little change, and although the tubular ducts may be separated by collections of inflammatory cells with germinal centres, the epithelial cells may appear quite normal in shape and staining reaction. Stoerk speaks of islands of glands of the intestinal type occurring in the secreting layer in gastritis cases.

Such islands have not been found in the series of cases here reported. In view of the hyperacidity so often found in gastritis it is interesting to note that neither hypertrophy nor hyperplasia of the cells secreting the gastric juice has been observed in the sections. Atrophy of the secretory epithelium is, however, a common finding. In well-established cases areas are often seen where the gland cells are practically absent, the whole mucous membrane being replaced by inflammatory tissue, cysts, and the remains of the glands and ducts, with or without follicles and germinal centres.

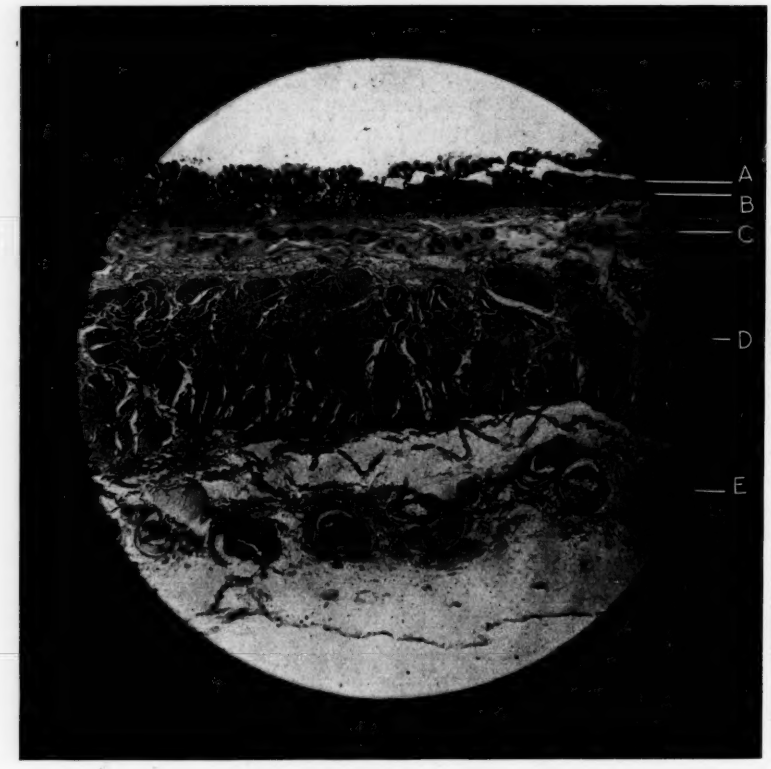

FIG. 21.—Chronic follicular gastritis, advanced stage. A, Mucous membrane densely infiltrated with inflammatory cells, and showing lymph follicles with germinal centres; B, Thickened muscularis mucosæ; C, Fibrosis of submucous area and many dilated blood-vessels; D, Normal muscularis propria; E, Dilated blood-vessels in the subserous layer.

It is suggested by Faber that achylia gastrica may result from the extensive atrophy of the secreting elements in continued gastritis. While this may be true, it is quite certain the achlorhydria may occur in cases where very little atrophy can be found in the sections. In two of the cases here reported (*Cases* 1 and 7) hydrochloric acid was not detected in the stomach contents, but the glands showed little atrophy under the microscope.

Thickening of the muscularis mucosæ is a constant finding in chronic follicular gastritis. Instead of being a thin compact sheet of muscle tissue this structure becomes thickened by hypertrophy, hyperplasia, and fibrosis. It may also be split into layers, between which are seen the same types of

inflammatory cells as are found in the mucosa. Beneath the muscularis mucosæ the inflammatory cellular exudate is much less pronounced. Islands of round cells, polymorphs, and eosinophils are commonly found at intervals in the submucosa, between the muscle layers of the muscularis propria, and even in the subserosa layer, but they are not always present. A small blood-vessel is generally to be found in the neighbourhood of each collection of cells. The small aggregations of cells in the subserosa probably precede the formation of the perigastric adhesions so frequently found at operation.

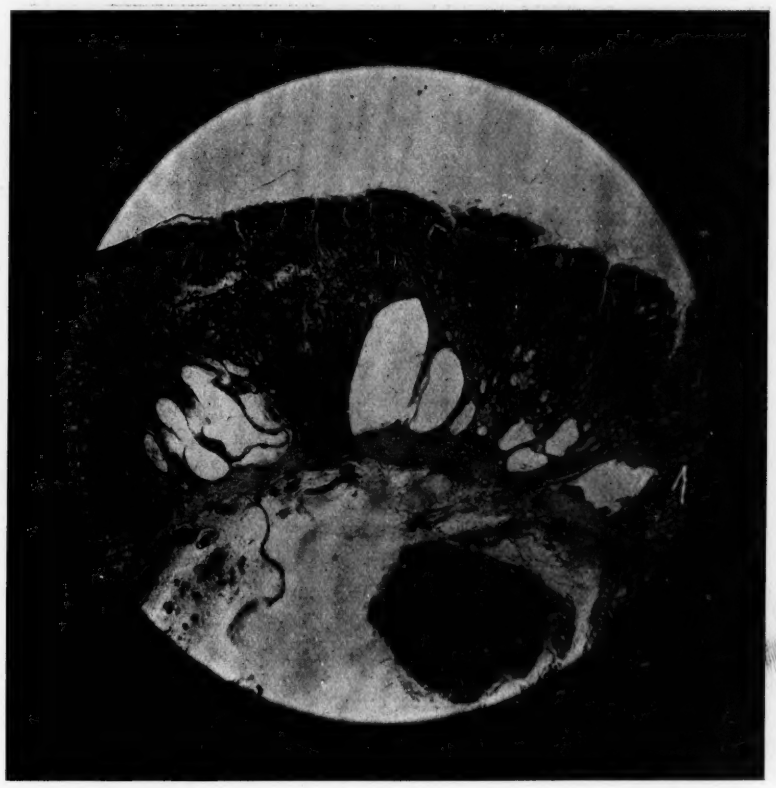

FIG. 22.—Chronic follicular gastritis, late stage, showing atrophy and cyst formation in gastric mucous membrane.

A fairly constant and striking feature of the lesion is the apparent increase in number and the distension of blood-vessels in the submucous and subserous layers. The small blood-channels of the mucous membrane itself are also frequently very prominent. These vascular features help to explain not only the hyperæmia of the peritoneal surface observed at operation, but also the occurrence of bleeding into the stomach. Areas of œdema in the muscularis propria are to be recognized in some of the specimens.

The small *hæmorrhagic erosions* of the mucous membrane are of interest in connection with the gastrostaxis that may accompany gastritis. Histologically, the erosions consist of narrow clefts which cut through the surface at right angles, and extend for varying distances into the mucous membrane.

They occur either at the summit of small elevations and lead down into lymph follicles or their neighbourhood, or they are found in deep clefts between rounded elevations of the mucosa and extend downward as deep as the muscularis mucosæ. Their edges are always sharp and well defined. Superficial necrosis in the walls of these clefts does not appear to be a prominent feature. In this series they have not been found to communicate with the lumen of blood-vessels. The general appearance of the erosions is that of relatively small and unimportant defects, but massive bleeding and even

FIG. 23.—Chronic follicular gastritis, showing superficial erosion of the mucous membrane, leading down to a lymph follicle.

fatal hæmorrhage have been reported. Superficial ulcerations of the uppermost layers of the mucosa may occur in the acute ulcerative form of gastritis.

A relationship between hæmorrhagic erosion or superficial ulcer on the one hand and chronic peptic (gastric) ulcer on the other has not yet been established beyond question. Aschoff, in his theory of " mechanical-functional disposition of the gastric pathway ", suggests the possibility of transition of erosions of the gastric street and isthmus ventriculi to true callous ulcer. Absolute proof of such an event is yet to be brought forward. That it is possible cannot be gainsaid, but that chronic ulcers invariably begin as erosions is quite another thesis, and must await proof.

Chronic duodenitis frequently accompanies chronic follicular gastritis.

There are some features in its gross and microscopic pathology which suggest a close relationship to gastritis, but for the purpose of the present study it is considered a separate though possibly an allied disease process.

COMPLICATIONS.

From the standpoint of pathological anatomy there are six chief complications or sequelæ of chronic gastritis—viz., hæmorrhage, pyloric stenosis, gastritis polyposa, atrophic pangastritis, perigastritis with adhesions, and cancer.

In the absence of accurate knowledge on the point, *hæmorrhage* into the gastric lumen is attributed to erosion or superficial ulceration of the mucosa.

Pyloric stenosis, with retention, frequently found clinically, may depend upon hypertrophy and hyperplasia of the muscle, pylorospasm, polypoid overgrowth of the mucous membrane, or a combination of any two or all three of these factors.

In *gastritis polyposa* the visible projections of gastric mucous membrane are composed of hyperplastic epithelial masses, or small adenomas with an epithelial covering. This form of gastritis is one of the best known, and diagnosis by X-ray is not difficult. Hæmorrhage, pyloric obstruction, and cancerous growth are its serious complications.

Atrophy of the mucosal cells can be found in almost every case of chronic follicular gastritis. In the acute forms, and in the ulcerating and bleeding types, it is not a prominent feature, but in the advanced cases it can nearly always be demonstrated. The atrophy is patchy in distribution, and cysts are present. Faber teaches that the natural course of gastritis is first hypersecretion, later gradual atrophy, finally achylia. As mentioned above, it is not necessary for the whole mucous membrane to be atrophic before achylia sets in, but, on the other hand, when achylia develops it is certain that a very considerable part of the secreting tissue has been destroyed. In late cases the whole stomach shows atrophy (pangastritis). The disease is not then confined to the pylorus, as in follicular gastritis, but involves the whole organ. As atrophy proceeds achylia becomes more pronounced. Free acid disappears first from the stomach contents, then pepsin, and finally rennin. From a clinical point of view a cure is possible as long as rennin can be detected in the gastric juice.

In four of the nine cases here reported *perigastric adhesions* were found (perigastritis adhesiva). They were most prominent about the pylorus in all cases, but in one (*Case* 8) the duodenum also showed organized adhesions to the surrounding structures. The microscopic sections from *Case* 3 are instructive in this connection. They show definite nests of inflammatory cells (polymorphs and round cells) between the strata of the muscular coat, and even in the subserosa. The inflammatory process may apparently penetrate the gastric wall, and lead to localized peritonitis resulting in adhesions.

DIAGNOSIS, PROGNOSIS, AND TREATMENT.

Diagnosis.—The presence of chronic follicular gastritis is to be suspected in cases with a long clinical history of indigestion accompanied by hæmorrhage, if no ulcer or cancer can be demonstrated by the X rays. If there

is hyperacidity in the early stages, or anacidity later, the suspicion is strengthened. A slight retention makes the diagnosis fairly certain. Temporary improvement under conservative measures, with sudden recurrence of bleeding or bouts of pain, serve further to confirm the opinion. The diagnosis is made fairly certain if inspection of the stomach after the abdomen is opened reveals perigastric adhesions, reddening of the serous coat, hardening and thickening of the walls in the pyloric region, or enlarged soft lymph-glands. A positive diagnosis, however, can only be made with the aid of the microscope.

DIFFERENTIAL DIAGNOSIS.—

1. *Chronic gastric ulcer* is differentiated from chronic follicular gastritis by the presence of epigastric tenderness, and by craters in the X-ray films.

2. *Gastric cancer* will nearly always be detected by X rays. Konjetzny's two cases of incipient cancer in the mucosa of patients suffering from chronic follicular gastritis are strong evidence that cachexia of sudden onset in such cases should be interpreted as heralding the carcinomatous change, and treated accordingly.

3. *Œsophageal varix* may be ruled out by consideration of the clinical history, and by abdominal examination.

Prognosis.—It is quite possible that the disease may exist for decades. The usual expectation is that it will last for many years unless hæmorrhage, cancer, chronic ulcer, pyloric obstruction, or achylia should modify the course. The underlying pathology is chronic inflammation, and there is an undetermined psycho-nervous element superadded. Psychic effects (e.g., distaste for work, headache, loss of sleep, vague indigestion, or actual abdominal pain) play an important and often predominant part in the clinical picture.

Treatment.—

CONSERVATIVE TREATMENT.—When this is attempted it is carried out on a symptomatic basis—for example, bland diet, alkali for hyperacidity, hydrochloric acid for anacidity, gastric lavage for retention, bitter tonics for anorexia, belladonna for pyloric spasm, and purgatives if necessary for constipation.

OPERATIVE TREATMENT.—It goes without saying that, in *secondary gastritis*, the primary lesions should always be treated. The secondary gastritis may accompany chronic ulcer, cancer, neurasthenia, syphilis, hernia, gastroptosis, pylorospasm, tabes, pulmonary tuberculosis, neurosis, perigastritis, previous gastro-enterostomy or partial gastrectomy.

Without entering into an argument of conservative treatment *v.* operative treatment, suffice it to say that our present knowledge is built up from operative cases, the data of which are complete and available. The case in favour of conservative treatment is yet to be made. Wanke treated by conservative measures 22 cases in which a normal-appearing stomach had been found at operation in cases of gastritis: 4 were cured (19 per cent), 8 were improved (36 per cent), and 10 failed (45 per cent).

In *primary gastritis* gastro-enterostomy has few supporters. In two cases in the present series this operation failed, and subsequently pylorectomy had to be undertaken to effect a cure.

Extramucosal pyloromyotomy, with which the name of Payr is associated, was introduced by him for treatment of those cases without ulcer which

showed true pylorospasm, with or without gastrostaxis or delayed emptying. When used in 16 selected cases (Payr, 1925), 12 were very well or fairly well, 2 improved, 2 not improved; 1 case died of phlegmonous gastritis.

Resection of the pyloric portion of the stomach and first part of the duodenum is supported by Konjetzny, Finsterer, Flörcken, Elter, and Nicolaysen (*Table II*). The continuity of the tract is re-established by

Table II.—Results of Partial Gastrectomy in Chronic Gastritis.

Name	Date	Interval since Operation	Total	Cured	Improved	Failed
Elter	1925	Over 1 yr.	15	12	2	1
Wanke	1929	Up to 4 yrs.	13	5	2	6
Nicolaysen ..	1928	1 yr.	7	7	—	—
Finsterer	1924	—	35	35	—	—
Paaby	1927	—	3	3	—	—
Konjetzny ..	1924	6 wks. to 10 mos.	8	8	—	—
This series ..	1930	6 wks. to 29 mos.	9	7	2	—
		Totals ..	90	77	6	7

Billroth I, or by the Polyá, Moynihan, Balfour, or Hoffmeister modifications of the Billroth II procedure. Wanke gives a warning that cases resected early in the disease are more likely to have post-operative trouble (fundus gastritis) than those operated upon late, when the inflammation is confined to the pyloric region. In the 9 cases of this series the operation known as the Polyá modification of the Billroth II procedure was carried out in 6 cases; in 2 the pyloric portion of the stomach and the first part of the duodenum were removed, (pylorectomy), leaving intact a previously existing gastro-enterostomy; in 1 case the Balfour method of partial gastrectomy was employed (*Table III*).

Table III.—Results of Resections and of Pylorectomy in Chronic Gastritis in the Present Series.

Operation	Total	Cured	Improved
Partial gastrectomy	7	5	2
Pylorectomy ..	2	2	—
Totals ..	9	7	2

SUMMARY.

In the nine cases of chronic follicular gastritis reported above there were symptoms or signs suggesting chronic ulcer, but no ulcer was found at operation or in the gross specimen. In all cases histological study of the portion of the stomach removed at operation showed a characteristic lesion, of which the most prominent finding was infiltration of the mucous membrane by inflammatory cells, in places forming large *follicles with germinal centres.*

Critical study of the results of partial gastrectomy in the present series and in the cases in the literature shows that the operation cures or gives relief in most cases, and fails only in a small number. Until the etiology is known partial gastrectomy (Billroth I or modified Billroth II) should be the treatment of choice when conservative measures fail. Pyloromyotomy probably has a place in cases where pylorospasm is present, although it will not guarantee freedom from the possibility of subsequent hæmorrhage or cancer.

This study was carried out in the Department of Pathology of the Montreal General Hospital, under the direction of Dr. L. J. Rhea, and the cases are reported by the kind permission of Dr. A. T. Bazin, Dr. E. M. Eberts, Dr. W. L. Barlow, Dr. C. K. P. Henry, and Dr. A. Stewart, of the Surgical Staff.

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CHONDROSARCOMA OF THE FIBULA.

By A. VICTOR NEALE and E. D. ALLEN-PRICE, Birmingham.

Ewing[1] states that the cellular chondromas are comparatively benign neoplasms, and conservative local treatment often proves satisfactory. Osteoid chondroma is a peculiar form of skeletal chondroma arising beneath the periosteum of long bones. As the growth enlarges, the bone assumes a thickened spindle outline simulating in form some of the osteosarcomas. A tumour which grows very slowly is probably of low malignancy, but may change its character and grow rapidly. Slowly growing local tumours may produce metastases early. Encapsulation is an important factor in the prognosis of the sarcomas occurring in relation to bones. Phemister[2] has recently recorded some observations concerning chondrosarcoma, and he is inclined to place this type of neoplasm in a separate group. Cartilage may be seen in ossifying sarcoma of bone, while the tumour may contain cartilage alone and show no metamorphosis.

Simple chondromas which originate on the surface of a bone or beneath the cortex and subsequently break through to the surface are likely to possess islands and branching areas of calcification. A case of this nature is recorded by Phemister :—

Male, age 14. Large swelling in the upper portion of the right leg which had developed for nine months with increase of pain. Physical examination was otherwise negative and the chest clear. X rays showed a large oval swelling in the upper seven inches of the right tibia. At operation the femoral vein was found to contain a column of cartilage, and microscopically the tumour consisted of hyaline cartilage with extensive areas of necrotic degeneration. Nine months later the boy was in excellent health.

True chondrosarcoma consists of islands of hyaline cartilage, areas rich in mitotic figures, and older calcified or ossified portions, the calcification being irregular and blotchy, giving a characteristic radiographic picture. Usually metastases occur later than with other types of sarcoma ; there is a tendency to invade veins, and metastases are cartilaginous. Phemister records that out of 61 cases of bone sarcoma, 10 were chondrosarcoma, the tumour having occurred in the femur, humerus, tibia, maxilla, spine, or rib. No recorded instance has been found of this type of tumour occurring in the fibula.

Chondrosarcoma may arise centrally or peripherally. The central type causes reactionary bone to form around, whereas the peripheral type does not cause bone reaction but gives a typical X-ray picture of a tumour with scattered areas of calcification.

Details of an interesting case of chondrosarcoma are given by Phemister :—

Male, age 40. The patient had noticed an outgrowth on the lateral aspect of the right tibia for about thirteen years. At the age of 27 this mass began to increase

in size, and at 34 it was as large as a fist. The neoplasm was excised locally, but it recurred, and at the age of 37 further operations were performed. Microscopically this tumour at the first operation consisted of apparently innocent cartilage. The tumour recurred again twice locally, and amputation was eventually necessary. Finally the tumour was found to be richly cellular, with karyokinetic figures very suggestive of malignancy.

This case resembles the one observed by us in the apparent innocency of the primary tumour and recurrence in malignant form after removal.

HISTORY.—A boy, age 16 years, stated that three years previously he was bitten by a gnat on the outer side of the right leg. A swelling developed here, and exercise

FIG. 24.—Radiogram taken in 1927, showing the calcific changes in the neoplasm.

FIG. 25.—Radiogram taken in 1930, showing the more diffuse nature of the tumour.

caused considerable pain. The swelling and pain increased and brought him to hospital.

ON EXAMINATION.—He was a healthy-looking boy and nothing abnormal was found except a lump on the outer side of the right leg. The lump was very hard and fixed to the fibula. It was 3½ in. long and 2 in. broad, with the centre at the junction of the middle and lower thirds of the fibula. Pressure on the lump, and flexion and extension of the ankle-joint, caused pain. He stated that recently the pain had been continuous and worse at night.

X rays (*Fig.* 24) showed that the tumour was on the surface of the fibula and

contained calcified areas. Its general appearance suggested malignancy. Radiographs of the chest showed no signs of metastasis.

FIRST OPERATION.—Local removal of the tumour was carried out in November, 1927. When exposed it appeared to be encapsuled. The tumour, together with the shaft of the fibula, was excised, the division of the latter being made 2 in. above and below the extremities of the growth.

SUBSEQUENT PROGRESS.—For two and a half years the patient remained well, led a normal active life, and grew 6 in. in height. In April, 1930, he again noticed a swelling at the site of the operation. This increased in size, and in August, 1930, after a trivial injury, continuous pain recommenced and he returned to hospital. A tender swelling in the original situation was present, and X rays revealed active sarcoma occupying the gap between the fragments of the fibula (*Fig.* 25). A Wassermann test was negative, and no metastases could be found.

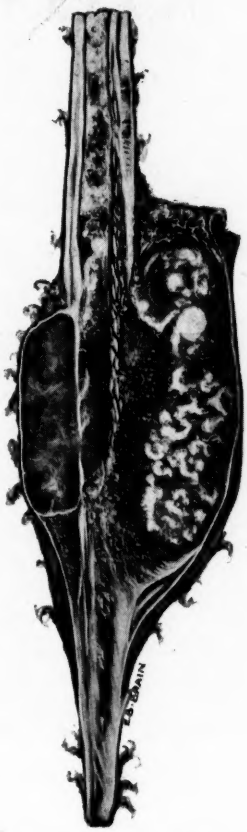


FIG. 26.—Tumour removed at first operation (November, 1927).

FIG. 27.—Recurrent tumour as seen at second operation (September, 1930).

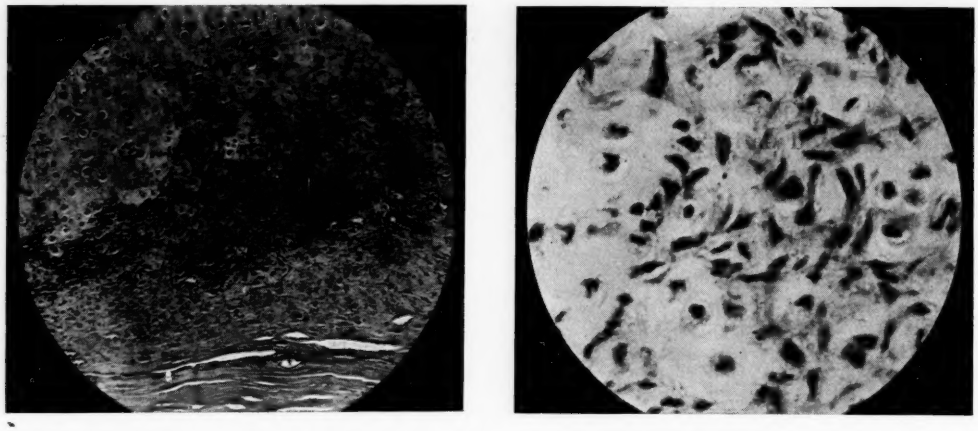
SECOND OPERATION.—Amputation through the lower part of the thigh was performed. Three months later the boy was in excellent health with no signs of metastasis.

PATHOLOGICAL REPORTS (*Figs.* 26, 27).—

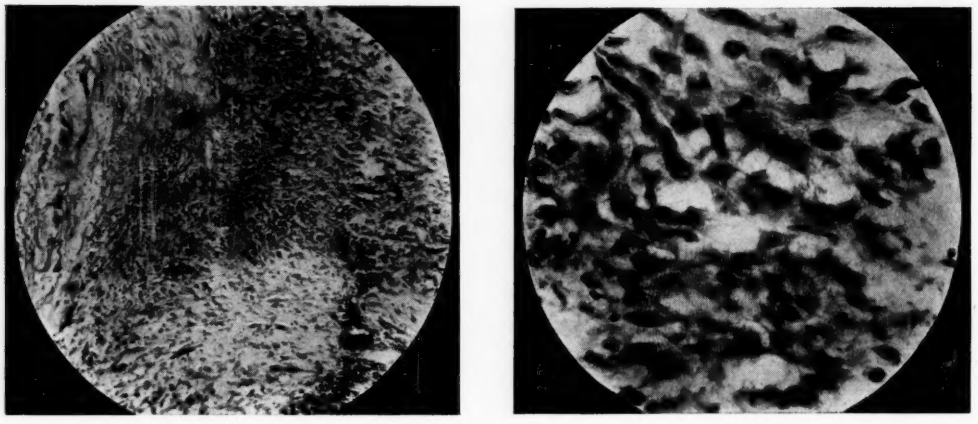
1. The tumour removed in 1927 was cylindrical in shape and had an indefinite fibrous capsule. It was firmly attached to the fibula and cartilaginous in structure.

Here and there was evidence of commencing penetration of the cortex of the bone, and, microscopically, in places there were areas of cell activity suggestive of early malignancy.

2. The recurrent tumour was an obvious chondrosarcoma, with definite cell activity and invasive character. The cells showed many mitotic figures, increased chromatism, and great variability in outline. Careful dissection did not reveal any infiltration of the soft tissues, and it still appeared to be confined to the bony structures.



FIGS. 28, 29.—Low- and high-power views of tumour removed in November, 1927.



FIGS. 30, 31.—Low- and high-power views of recurrent tumour removed in September, 1930.

An interesting feature of the case was the discovery of very early malignant changes in the first tumour and their more marked character in the recurring one. The essential origin of the tumour in cartilage is shown in the microphotographs (*Figs.* 28–31).

This case, like Phemister's, illustrates that sarcomatous change in a chondroma may be very gradual and probably remain within the bounds of cure by amputation for a moderate period of time (apparently about three years in the cases observed).

Chondrosarcoma may, it appears, remain amongst the less malignant sarcomatous tumours of bones, but no doubt too great a latitude cannot be allowed, because rapidly increasing infiltrative and metastatic capacities may arise at any time. It would probably have been a safer procedure, in the case outlined above, to have carried out amputation in the first instance. The very unusual situation of the neoplasm—namely, in the fibula, a site apparently hitherto unobserved—is of additional interest.

This case was under the care of Mr. Bertram Lloyd in the wards of the Queen's Hospital, Birmingham, and we are grateful to him for permission to record these observations. The microphotographs were kindly supplied by Dr. J. F. D. Shrewsbury.

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THE TIME FACTOR DURING WHICH VASCULAR RESPONSES PERSIST FOLLOWING PERI-ARTERIAL SYMPATHECTOMY.*

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A CURIOUS feature of the vascular responses to extirpation of the sympathetic peri-arterial network is their transient nature. In experiments designed to ascertain what vascular changes, if any, follow this operation, Hemingway and I have shown that in the cat and the rabbit peri-arterial sympathetic destruction increases local circulation, but that its effects are exceedingly transient.[1] A readjustment, the mechanism of which is at present obscure, comes into action at a time which is variable according to species and possibly also the vessel selected and the site of the particular vessel operated upon—for example, we found the readjustment in the case of the femoral artery of the cat to take place in a matter of seconds, whereas that following extirpation of the nerve net on the carotid of the rabbit persisted for some hours or days. This readjustment appears to follow also extirpation operations on the main sympathetic system. Thus we have noticed that vasodilatation in the ear of the albino rabbit consequent upon cervical sympathetic neurectomy is slowly recovered from, but have also observed that its effects are both greater and more lasting than the peri-arterial operation performed on the carotid artery.

A recent case throws some light on the probable duration of the time factor in man :—

HISTORY.—The patient was a steel worker, age 61, who had been an international footballer and was a healthy man with a comparatively healthy vascular system and a negative blood Wassermann reaction. Just over five years before admission his left leg had been severely scalded with hot paraffin. The leg ulcerated and had never healed since. For one year he attended three times a week for dressings at a hospital out-patient department. He had had long periods of rest, but with very little effect.

ON EXAMINATION.—Two ulcers were seen, one on the anterior surface of the leg in the middle third, the other, slightly larger, on the calf. The leg was discoloured brown, and the great toe showed onychogryposis.

OPERATION (Dec. 4, 1930).—Under chloroform and ether anæsthesia, the left femoral artery was exposed in Hunter's canal and found to be a healthy vessel. It was accompanied by two large nerves regarded as the internal saphenous and the nerve to the vastus internus, each as large as a normal ulnar nerve. These nerves were not lobulated, but uniformly enlarged, and appeared to be merely hypertrophied. The phenomenon was unique in the experience of those present in the theatre. The artery was stripped of its adventitia over a length of half an inch, and over this area and for a further quarter of an inch above and below was well

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painted with strong carbolic acid, a method which we had previously applied effectively and without detriment to the artery in animals. The deep fascia was closed with catgut and the skin with clips.

AFTER-HISTORY.—On the morning following the operation the left foot—that is the one on the side of the denervated femoral artery—seemed distinctly warmer to the touch than the right, and the patient complained of his right toes being cold while those of the left foot were warm. The epidermis of the whole of the leg next began to break up into scales, which gradually fell off, and the ulcers commenced to heal. By the thirty-ninth day the ulcer on the front of the leg had soundly healed and the whole leg had peeled, leaving healthy skin beneath. The ulcer on the calf had almost healed, but now seemed to be stationary. By the forty-fourth day the calf ulcer had not only ceased to heal but was definitely getting larger.* The anterior ulcer remained healed.

It thus appeared as if a falling off in the beneficial effects which were observed almost immediately to follow the operation took place somewhere about five weeks subsequent to the operation.

COMMENTARY.

The effects of the peri-arterial sympathectomy — namely, increased warmth, the scaling, and the reparative changes in the ulcers—were regarded as expressions of an increase in local circulation, the scaling being looked upon as due to proliferation and renewal of the stratum corneum consequent upon increased activity of the stratum cylindricum of the epidermis. As estimated by the condition of the ulcers, the response persisted for approximately five weeks, after which time its effects were no longer apparent. It would appear, therefore, that in man, as in animals, an increase in local circulation follows peri-arterial sympathectomy applied to the femoral artery, but that the increase is likewise a transient condition, lasting, however, for approximately five weeks. This period contrasts with those of a few seconds to as many days observed by Hemingway and the writer in plethysmographic and other experiments carried out upon cats and rabbits, and the wide variation in the length of the period in different species recalls the wide range of the duration of Monakow's diaschisis in animals and in man. It is regretted that a series of cases such as that from which these observations have been made is not to hand to enable a more precise and average measurement of the time interval to be made, but it is not often that cases as suitable as that here detailed present themselves for study subsequent to the operation, the majority either being discharged from hospital or coming to amputation of the limb before the post-operative reaction phase has terminated.

From observations made by the use of Pachon's oscillo-sphygmomanometer on cases in his clinic, Professor René Leriche[2] gave the writer to understand that he believes that the vasodilatation lasts for somewhere about the same time as that observed on purely clinical grounds in the case here recorded, but Dr. Leriche commented upon the difficulty of satisfactorily measuring vascular responses except by plethysmography. It may be concluded by analogy with the experimental work previously referred to that a

* By skin-grafting, this ulcer subsequently healed and the patient was discharged. He was seen a fortnight after his discharge, and had a soundly healed leg, but this is irrelevant as far as the thesis of this paper is concerned.

greater and more lasting increase in local circulation follows the extirpation of the sympathetic supply to the limb (by ramisectomy or ganglionectomy), but that from the observations here recorded, a vasodilatation lasting somewhere about five weeks will follow a successful peri-arterial sympathectomy applied to a healthy artery supplying a healthy capillary bed, and that in certain cases in which a transient vasodilatation of this duration will be beneficial, the operation may with advantage be considered.

The writer is much indebted to the Medical Research Council for partly defraying the expenses in connection with the experimental work referred to in this note.

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PAPILLIFEROUS CYSTOMA OF THE PETROUS BONE ASSOCIATED WITH HYPERNEPHROMA AND CYSTIC PANCREAS.*

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In a monograph on cerebellar cysts, Lindau[1] (1926) drew attention to the not infrequent coexistence of hæmangioblastomas of the central nervous system, especially of the cerebellum, with retinal angioma (von Hippel's disease); but in addition to this there have appeared fairly commonly associated lesions in other parts of the body. Thus in his series of 40 cases Lindau reported cystic pancreas in 8, renal cysts in 10, and hypernephroma in 6; cystic epididymis has also been noted. In the case we have to report the main lesions were a papilliferous cystoma of the right petrous bone, hypernephroma of the right kidney, cystic pancreas, and cysts in the right epididymis. While the tumour of the petrous bone was intrinsically interesting—so far as we can discover no exactly similar growth in this situation has been recorded—the associated lesions call for some comment, especially because there is a tendency to regard cystic pancreas as a pathognomonic feature of Lindau's disease.

CASE REPORT.

Clinical History.—J. B., male, age 23 years, was admitted to the Neurological Surgical Service of the Manchester Royal Infirmary at the instance of our colleague, Professor Tylecote, with the diagnosis of intracranial tumour, probably acoustic neuroma. Some twelve months before admission the patient had developed a slight weakness of the right facial musculature, which was the only symptom until, some months later, hearing became less acute in the right ear. Both of these disabilities steadily but slowly progressed, and on admission (July 5, 1929) a complete right facial paralysis was manifest, and deafness in the right ear, almost but not quite complete. During the previous two months headache was intense, and latterly had been accompanied by vomiting of such a degree as to cause great emaciation. The patient had become dizzy, and reeled to the right when he walked; on one or two occasions he had fallen to the right.

On Examination.—The patient presented the following signs: (1) Right facial paralysis, complete peripheral type; (2) Deafness in right ear with an unperforated tympanum; (3) Slight choke of the right optic nerve-head, venous congestion only on the left, no retinal angioma seen; (4) Nystagmus on fixation to the right; (5) No clear evidence in the limb musculature of cerebellar involvement. The subsequent autopsy showed that the cerebellum was neither infiltrated

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nor materially indented by the growth. The patient held his head flexed forward, and rotated slightly to the left. The diagnosis was not absolutely clear. The fact that the facial paralysis led in the symptomatology seemed to make it unlikely, but not impossible, that the tumour was an acoustic neuroma. We have had a series of these tumours in which deafness was not absolute, the tumour presumably originating in the sheath of the vestibular division, as Henschen contended. A basal endothelioma appeared more probable.

The question of treatment became one of some urgency because vomiting was now very distressing, and during the last few days there had been some difficulty in swallowing. At the same time the facial paralysis, the fixed posture of the head, the violence of the headache and vomiting, all combined to make a picture of unforgettable misery. The patient's skin was dry, and it seemed very unlikely that he would survive a lengthy or bloody operation.

Operation.—It was decided therefore to limit any immediate operative procedure to a decompression, with observation of the local condition, leaving the

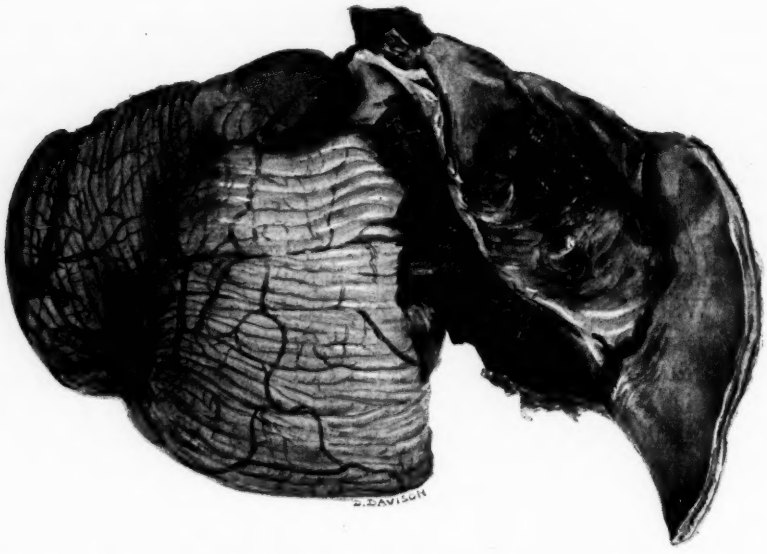

Fig. 32.—Tumour of right petrous bone seen from above, cerebellum retracted.

removal of the tumour for another session, unless it should promise to be unexpectedly easy. Accordingly on June 6 a wide suboccipital exploration was performed by the usual technique, very considerable foraminal herniation rendering laminectomy of the arch of the atlas imperative. The right cerebellar hemisphere was gently retracted and the vagal and facial exits were inspected. Dark-red, hard tumour with no clear boundary was encountered on the back of the petrous bone, and nothing further was done. The wound was closed in the usual careful fashion. The patient's condition was unaltered by this intervention; he did not cease to vomit, and despite the intravenous administration of glucose and hypertonic saline, he died of pneumonia five days after the operation.

Autopsy Findings.—The anatomical diagnosis was: Tumour of the right petrous bone indenting cerebellum but not invading it (*Fig.* 32); cysts in the pancreas; encapsulated tumour of right kidney; cysts in epididymis; bilateral bronchopneumonia. The pancreas contained a few cysts 0·5 to 2 cm. in diameter. The right kidney appeared normal in general aspect, but on the middle of its outer border there was a round encapsulated growth, 9 mm. in diameter, projecting under the renal capsule, which on section showed yellow patches alternating with red. In

the right epididymis there were a few hard cysts 0·75 cm. in diameter around the globus major, and a similar hard mass in the spermatic cord above. The vertex of the skull was normal. The dura was normal as seen from above. The pia and arachnoid were moderately congested. On removing the brain a nodulated dark-coloured tumour was seen beneath the dura covering the right petrous bone, the membrane being much raised and invaded. The right temporosphenoidal lobe was lightly adherent at one point about the tegmen tympani. The main body of the growth was seen to occupy the centre of the petrous bone, a fairly large extension passing upwards through the region of the tegmen. Similar extensions passed backwards and inwards from the region of the internal auditory meatus. The latter extension came into close relation with the cerebellum and pons in the region of the auditory nerve, but the relation to the nerve itself was not clear. On section through a portion of the tumour, its substance was found to be very dark in colour, and there was an appearance of numerous minute cavities filled by dark material. This suggested to the naked eye that the growth might possibly be an angioma.

There was no gross abnormality of the pericardium, heart, œsophagus, thyroid, stomach, liver, spleen, testes, or left kidney.

Dissection of the Right Temporal Region.—On drilling through the cortex covering the mastoid air-cells, a large isolated air-cell 1·5 by 1 cm. was soon entered. It was entirely uninvaded by tumour. The rest of the mastoid air-cells, and the mastoid antrum, were filled by gelatinous-looking tumour material. The bone forming the anterior wall of the external auditory meatus was nibbled away with forceps until the tympanic membrane was displayed. This was dark in colour and not obviously perforated. Certainly no tumour extruded through it. On removing further bone, the specimen could be opened in a horizontal direction into an upper and lower half, and the tympanic cavity was seen to be entirely filled by tumour. The growth could be easily separated from the tympanic membrane, in which two or three minute openings were seen after its removal. There seemed to be little doubt, however, that the tumour was not intimately connected with the membrane, because of the ease with which they were separated. We received the impression that the tumour had not arisen from the walls of the middle ear and that such erosion as was present was of a secondary nature. If it had originated here, the structures of the cavity would have been more profoundly altered. The tumour occupying the middle ear, the tympanic membrane, the malleus, and a specimen consisting of a small group of mastoid air-cells, were preserved for histological examination. The internal ear was then removed as far as possible *en bloc* for decalcification. The cut passed through the external semicircular canal, whence fluid exuded. No gross tumour material was seen, the internal ear giving the impression before it was opened of being free from growth. In general, the whole petrous bone, as far as its tip, seemed to be infiltrated. The carotid canal was not involved, and the artery was normal.

Histological Examination.—On histological examination tumour tissue was found in the mastoid air-cells, the internal ear, the tympanic cavity, and the malleus. The tympanic membrane was uninvaded by tumour. The tumour presented the same general appearance in these various sites, and may be described as a papilliferous cystoma.

The cysts were closely packed and irregular in shape, varying from 0·8 to 5 mm. in diameter. They were bounded by a single layer of flat to low columnar cells. The fibrocellular stroma supporting this layer of cells varied much in amount, in some areas being very scanty, in others abundant and dura-like (*Fig.* 33). In the dense parts of the stroma there were a few globular laminated concretions staining darkly with hæmalum, and occasional deposits of cholesterin. The cysts were filled with homogeneous hyaline material staining deeply with eosin, in which mucin could not be demonstrated by staining with mucicarmine, thionin, or methylene blue. Areas of hæmorrhage were visible towards the walls of many of the cysts, being on the whole most marked in cysts in which papillary ingrowths were few or absent. Groups of round cells, about 25 μ in diameter, laden with brown pigment, were seen in these hæmorrhagic areas.

The papillary ingrowths lay embedded in the hyaline contents of the cysts (*Fig.* 34). In the specimen from the middle ear the papillæ were so abundant that

FIG. 33.—Tumour of right petrous bone, showing dense fibrous stroma surrounding the cysts, in one of which papillæ are visible. (× 50.)

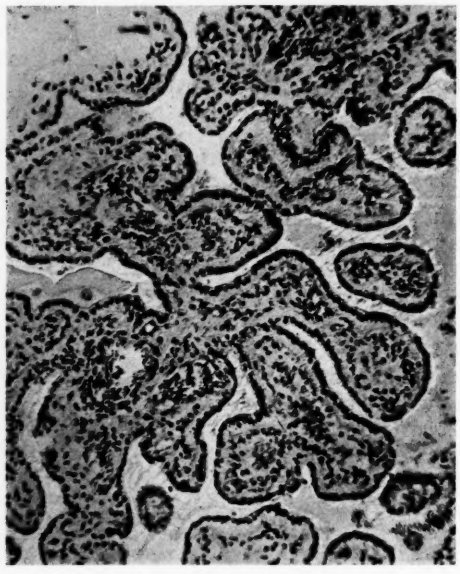

FIG. 34.—Tumour of right petrous bone, showing branching papillæ embedded in hyaline material. (× 100.)

FIG. 35.—Tumour of right petrous bone, showing part of a papilla whose covering cells have nuclei at their free poles. The vascular stroma is clearly seen. (× 200.)

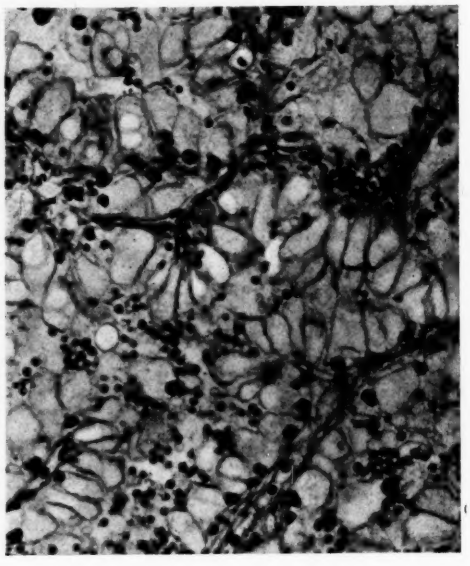

FIG. 36.—Tumour of right kidney, showing the sharp definition of the cells and their small round nuclei. (× 200.)

the cystic structure of the tumour was almost masked, the hyaline material being very scanty, though showing the features described where it could be found. The

papillæ were composed of a core of branching fibrocellular stroma continuous with that surrounding the cysts, but relatively more vascular, covered by a single layer of low—rarely tall—columnar cells. Direct continuity between the cells covering the papillæ and those lining the cysts was established in places. The cytoplasm of the cells covering the papillæ was clear, and their nuclei large, oval, or cuboidal in shape, and containing a moderate amount of chromatin. The nuclei often lay at the free border of the cells, giving the growth a striking appearance (*Fig.* 35). This feature was best seen where the cells were tallest. In some of the cells appropriate examination showed the presence of fat and doubly-refracting bodies, which occurred irregularly in scattered patches. The specimen of the internal ear and malleus showed invasion and destruction of the bone, the tumour itself retaining its characteristic morphology. The peripheral nuclei of tall columnar cells forming the covering of the papillæ were particularly well seen in the internal ear. It was possible to recognize the organ of Corti in places.

The tumour of the right kidney was encapsulated by fibrous connective tissue. Within this were enclosed acini and solid alveoli of epithelial cells supported by a scanty delicate vascular stroma (*Fig.* 36). The cells were fairly large, of cuboidal or low columnar shape, with sharply defined walls, small round nuclei, and clear bodies. By suitable staining they were found to contain abundant neutral fat, some doubly-refracting fat, and abundant glycogen. The growth corresponded in structure with the Grawitz tumour of the kidney or hypernephroma, but there was no evidence to show that it had penetrated its capsule or that it was in active growth.

The cysts in the pancreas were lined by a single layer of cuboidal cells with spherical nuclei. Careful examination failed to demonstrate any neoplastic process in these cysts.

DISCUSSION.

Objection may be taken to our use of the term 'cystoma', admittedly a purely morphological designation. The cells composing this tumour were clearly epithelial, but of such peculiar type that it is impossible to be certain exactly whence they had been derived. The tumour was composed of fully differentiated elements, and was structurally uniform. The epithelial cells, while varying much in height from place to place, were individually well defined units. Indications that the tumour was not recently formed were the presence of concretions, old hæmorrhages, sporadic deposits of cholesterin, and the fact that the fibrous stroma showed a marked preponderance of fibres over cells.

We are satisfied that the histological appearances definitely exclude an etiological relationship between the tumour of the petrous bone and that of the kidney. Each had a fairly uniform and distinctive structure, and no transitional stages between them could be found. The cystic structure, the hyaline material, the papillæ embedded in it, with their remarkable cubical or columnar epithelium with peripherally situated nuclei, and the dense stroma with concretions, were features peculiar to the petrous tumour. The renal tumour, on the other hand, presented a picture conforming in every way with the Grawitz tumour. Its acinar structure with solid alveoli of sharply defined cells containing small dot-like nuclei irregularly arranged, its uniformly delicate vascular stroma, its abundant glycogen, and its well-formed fibrous capsule, were without counterpart in the petrous tumour. Whereas there was no evidence of active growth in the renal tumour, the petrous tumour was seen to have invaded and destroyed bone.

In considering the possible intra-petrous sources of such a tumour as this, we have been led to review those recorded cases of primary carcinoma of the middle and internal ear whose structure in any way approached that of our own. Such cases are few, the most notable being those of Lange,[2] Furstenberg,[3] and Maestranzi.[4]

Lange[2] (1904) was the first to record a case of cylinder-celled carcinoma primary in the middle ear. The external auditory meatus was occupied by a polypoid growth that was firmly attached to the tympanic membrane but nowhere adherent to the meatal wall. The tumour caused widespread destruction of the petrous bone, and metastases were found in the regional lymph-glands, in those of the neck, and in both lungs. It was characterized by an abundant production of mucus as shown by metachromatic staining with a dilute thionin solution. Histologically it showed a simple epithelium composed mainly of columnar, partly of cubical, cells with basal nuclei, that formed the lining of spaces with mucous contents in which clumps of rounded cells lay free. The stroma was scanty. Calcium carbonate concretions were present in both primary and secondary growths. Accepted by Furstenberg[3] (1924) and Marx[5] (1926), the diagnosis was impugned by Schlittler[6] (1919), who pointed out that Lange failed to describe the histological structure of a submaxillary swelling noted at the autopsy, and that it is in consequence uncertain whether this swelling represented enlarged lymph-glands or a tumour of the submaxillary salivary gland. Schlittler maintained that the histological account of Lange's tumour was not incompatible with that of a deposit secondary to a tumour of the submaxillary salivary gland. That such tumours may be morphologically indistinguishable from carcinomas, and that they frequently exhibit degenerative changes of various kinds, has, according to Schlittler, been shown by Küttner.[7]

Furstenberg[3] described a primary adenocarcinoma of the middle ear and mastoid. The external auditory meatus was not affected, squamous epithelium being continuous over the tympanic membrane, which the growth had involved. The tumour was not uniform in structure, being alveolar in some parts and glandular in others. The glandular parts were very irregular. The abundant stroma gave the centre of the growth a scirrhous appearance. Warthin, who made the pathological examination, regarded the growth as primary in the middle ear, "representing a teratoid disturbance of development with blastomatous transformation".

Maestranzi[4] (1927) described a primary alveolar cancer of the middle ear. It presented as a polyp unattached to the wall of the external auditory meatus. Invested by stratified squamous epithelium, its substance contained alveolar cavities occupied by clumps of cells corresponding in type to cylindrical epithelium. Some cells lined the walls of the cavities, others lay free within them. In general appearance this tumour was not unlike that described by Furstenberg.

Of these tumours, the only one to which our petrous tumour bears any close resemblance is that of Lange. From this it differs both cytologically and histochemically, and also by the fact that it was the only growth of its kind in the body. From Lange's tumour, again, and also from Maestranzi's, it differs in that it it did not penetrate the tympanic membrane, a point

against the view that our tumour might have arisen in the tympanic cavity. If the growth began in the internal ear, complete destruction of the latter might have been expected, in view of the extent of the growth in other parts.

The peripherally placed nuclei of the taller epithelial cells of the papillæ suggest a thyroid or parathyroid origin. No tumour was found in these organs at the autopsy, but that the petrous tumour could have started from an embryonal rest is not entirely impossible in view of the close developmental relationship subsisting between thyroid, parathyroids, and middle ear. The possibility of Lange's tumour having originated either from embryonal rests or from a primary growth in the thyroid does not appear to have been considered by Lange or by Schlittler.

We have finally to consider whether our petrous tumour might have been derived from neural epiblast, a suggestion made attractive by the discovery in the patient of a series of congenital lesions identical with those described by Lindau as occurring with hæmangioblastomas of the central nervous system. Our petrous tumour bore no resemblance to a hæmangioblastoma, and Lindau, to whom we submitted the slides, was of the same opinion. He suggested that our tumour was possibly collateral to the hæmangioblastomas of the central nervous system, and that it might have arisen from the anlage of the choroidal system, derived from the neural crest; the companion lesions would fortify this suggestion.

Hence it is possible that the lesions in our case may be the result of an error in development cognate with that which must underlie the lesions correlated by Lindau. In cases exhibiting multiple congenital lesions any one of them might elaborate itself into the clinically dominant feature. Pathological interest, however, centres in the developmental error rather than in any individual lesion that may declare it. It seems not unlikely that Lindau's disease, and the case we have described, are simply special forms of a more general disorder.

Whether cysts of the pancreas can be regarded as presumptive evidence of one particular type of tumour is in our opinion debatable. A highly cystic pancreas has been figured independently by Hadfield[8] (1929) in a recent report on a cerebellar angioma. Lindau[9] (1931) states that he does not think that these associated somatic anomalies have an angiomatous origin. It remains to be seen whether future observations will confirm the particularity of this association. Our own case throws doubt on it, though it must be admitted that the pancreas itself was not exactly of the type that Lindau regards as pathognomonic of his syndrome. Had our patient survived long enough, there is the possibility that the pancreas would eventually have conformed to type.

From the clinical standpoint it is clear enough how the symptoms in the case we have described came to mimic a posterior fossa tumour, and a more accurate diagnosis might have been made had the skiagrams, which actually showed changes in the texture of the petrous bone, been better interpreted. The posture of the patient, so important clinically, served to deviate the head out of the true antero-posterior axis in the skiagrams, so that a failure of upper contour was missed. The pathological study of

the growth, however, showed that its true nature could not have been determined clinically. Only once have we seen a similar erosion, and that was due to a cavernous angioma.

In conclusion it is our pleasant duty to thank Professor Lange, of Leipzig, and Dr. Lindau, of Lund, for personal communications, and Professor J. Shaw Dunn for his valued interest and counsel during the course of this study.

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A CLINICAL STUDY OF ACUTE APPENDICITIS IN OLD AGE.

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It might well be supposed that the last word had been said on the subject of appendicitis, but during the past few years my attention has been drawn forcibly to a considerable number of cases of a uniform but very anomalous type occurring in people in advanced years. The picture presented is so constant that I find that such cases may be confidently diagnosed from the other conditions which they mimic. As they are not even mentioned in the text-books, I have ventured to present this short clinical study with the hope that it may be of some assistance to those who are confronted with similar cases. The anatomy and etiology of appendicitis will not be discussed, but consideration given only to the clinical manifestations of the disease which are peculiar to old age.

I am aware that any definition of old age must be, to a large extent, an arbitrary one. It is frequently stated that acute appendicitis is rare in old age, but in my own experience this is far from being the case, and though 60, which I make the lower limit, may seem somewhat drastic, it does include every case in my series, each of which is a manifestation of the declining power of the body to contend with bacterial infection. In the last three years I have had twelve such cases, in addition to at least as many more which presented symptoms comparable to those encountered in the large run of cases in young people. It is clear, therefore, that appendicitis in old people is far from being a rarity. Of my twelve cases, nine were men, and three were women, and their ages were: 62, 63, 64 (2 cases), 69 (2 cases), 71, 72, 73, 74, 75, and 86.

The underlying factor in all these cases is the alteration in the reaction of the tissues concomitant with old age. This alteration is general in all parts of the body, and is characterized by a tendency to chronicity following on a lengthened period of development. It is seen in both the reaction to infection, and—what is equally important—in the delay of healing processes. As a clinical illustration of the first, one may cite senile tuberculosis of the cervical glands, and of the second, the greatly increased time necessary to produce adhesion between two layers of peritoneum. It is, indeed, the behaviour of the peritoneum that accounts for many of the anomalous cases of appendicitis under consideration.

Before going more fully into the symptomatology of the clinical types, there are certain features which are common to nearly all cases. There is, first, a much longer history than is usually obtained, and it is not uncommon for seven or eight days to elapse before the symptoms become acute enough

to force the patient to seek surgical aid. This is in marked contrast to the customary twenty-four or forty-eight hour illness with which we are confronted. The average length of history in my cases was between five and six days. There is far less constitutional disturbance, and the amount of pyrexia is unusually small. Pain is comparatively less when due solely to the inflamed appendix, and when marked is the result of secondary peritonitis and distension. All visceral processes are considerably slowed, and constipation is the rule.

The majority of cases fall into a group presenting nearly all the signs and symptoms of acute or subacute intestinal obstruction, rather than an acute inflammatory lesion. There is a history of constipation lasting seven or eight days, the patient passing neither fæces nor flatus. Even more striking in this regard is the failure, often experienced, to produce any relief by repeated enemata, a circumstance strongly suggestive of an organic obstruction. Equally misleading is the history, sometimes given spontaneously by the patient, of increasing difficulty extending over a period of several months.

Vomiting is not a constant feature, but quite often occurs at the onset of the illness, subsiding completely by the time the patient is seen by the surgeon. This is rather in favour of an inflammatory as opposed to an obstructive lesion. The amount of pain varies, but is mostly the outcome of abdominal distension, and its distribution is a wide one, over the whole of the lower half of the abdomen. Not infrequently the pain and tenderness are most strongly marked in the left side, due largely to the upward displacement of the pelvic coils of distended ileum. The abdomen as a whole is markedly distended and resonant. The tongue is dry and furred, and the breath offensive. On rectal examination the bowel is found to be empty, and the frequent failure of enemata to bring relief shows that this emptiness extends far above the portion within reach of the examining finger. I cannot illustrate the condition better than by giving the notes of two actual cases which have come under my care.

Case 1.—Mrs. E. M., age 62, was admitted to the West Norfolk and King's Lynn General Hospital on March 15, 1927. On March 8 she began to have daily sickness, but vomited only small quantities. The sickness was associated with some abdominal pain. The bowels had been confined since the first day of the illness—that is, for seven days—and the day previous to admission two enemata had been given without result. On admission two more were given, and the second brought away only a few small fragments of hard fæces. She volunteered that she had noticed increasing constipation for six months. On examination she looked very ill. The tongue was dry and furred. The abdomen was much distended and uniformly tender and resonant over the whole lower half. The temperature varied between 99° and 100°.

A diagnosis of acute intestinal obstruction was made, and the abdomen opened by a mid-line incision. Peritonitis was found, and a gangrenous appendix, lying over the pelvic brim and quite free from adhesions, was removed. The abdomen was drained. Healing was delayed, but the patient recovered and was sent home well at the end of five weeks.

Here was a case with all the history, signs, and symptoms of acute intestinal obstruction in which an error of diagnosis might well be excused.

In the next case to be recorded, one was similarly misled, but fortunately the correct diagnosis was arrived at before operation.

Case 2.—Mr. J. S., age 75, was admitted on July 28, 1929. He gave the following history. Increasing constipation for six months without any diarrhœa or passage of blood. For four days previous to his admission he had absolute constipation accompanied by generalized abdominal pain. He had vomited once.

On examination he was a frail wasted old man. The abdomen was moderately distended and slightly tender over the lower half. His temperature never exceeded 99°. Enemata produced only small pieces of fæces and a little flatus. It was decided to operate on him as a case of intestinal obstruction, but on examination of the abdomen under the anæsthetic, a swelling low down in the right side, which could not be felt before, became apparent. A muscle-splitting incision was made, and an appendix abscess drained, the appendix itself being removed. He made an excellent recovery and was discharged at the end of three weeks.

The other cases in this group give very similar histories, but the feature of them all is absolute constipation for a varying number of days, associated with sickness and marked abdominal distension. What we are confronted with in reality is an acute infective process in which the local reaction is so slow and mild that by the time the patient seeks surgical aid the original disorder is masked almost entirely by a new one, which is no more than a complication of it. Doubtless if these cases were seen in the early stages, the difficulties of diagnosis would be far less. That they are not seen is only a reflection of the mildness of the initial symptoms.

The diagnosis of these cases is always difficult, but there are a few points which, borne in mind, may enable us to arrive at the correct conclusion. First of all, although apparently superfluous, I would suggest that acute appendicitis should be considered as a possibility in all cases of acute or subacute intestinal obstruction. Secondly, the distribution of pain and tenderness should not be allowed to influence the diagnosis too much. I have already mentioned that left-sided pain and tenderness sometimes are predominant. I should like to stress this point, as it is of frequent occurrence, and due, as I have repeatedly confirmed at operation, to distended and inflamed coils of ileum which have risen from the pelvis. In old people, therefore, we are faced with this seeming paradox, that in such cases of acute appendicitis the local reaction is comparatively so small that maximum pain and tenderness are left-sided and due to a secondary distension, whereas in cases of true organic obstruction in the large bowel, maximum pain and tenderness may be, and often are, right-sided, owing to the easily distended cæcum.

(Since commencing this article, I have been asked by a colleague to see a further case in which this situation confronted us, and in spite of marked left-sided pain and tenderness, and considering the history carefully, a diagnosis of appendicitis was made and confirmed at operation. There was no shutting off of the inflamed appendix, and the pelvic coils of ileum were distended and displaced upwards.)

A third point in the diagnosis is the presence of pyrexia, which, though comparatively little, is definitely in favour of an inflammatory lesion as opposed to a purely obstructive one. Vomiting is less marked than would be expected in an obstruction with the same degree of distension.

No single one of these signs is sufficient evidence on which to make a diagnosis, but by bearing them all in mind, I believe that in nearly all cases one can arrive at the right conclusion.

The intra-abdominal findings at operation are worth recording, as they shed considerable light on the peculiar signs and symptoms. I have already made some reference to the appendix itself. The most striking feature is the freedom from adhesion to surrounding structures and omentum, even after an illness of over a week. It may have a patchy covering of thin lymph, which spreads on to the cæcum and terminal coil of ileum, but there is no complete shutting off, so that a way is clear for the passage of pus and exudate into the pelvis and general peritoneal cavity.

Where peritonitis is present, the pus is often of an unusual nature, being very scanty and extremely greasy, yet forming a thin film over the whole pelvic contents. It is more common to find this condition than an excess of exudate. The pelvic coils of small intestine, as already noted, are injected and distended secondarily to pelvic peritonitis, but occasionally the distension may be partly due to a mechanical obstruction at the ileocæcal angle by attempts at forming adhesions in the vicinity of the primary infection. The distension of the ileum in these cases never seems to call for active relief, and subsides quite quickly when the abdomen is drained.

The treatment of these cases is always operative, and the abdomen should be opened by a mid-line incision. Owing to the absence of adhesion, the appendix can nearly always be delivered easily and safely. The peritoneal cavity should always be drained adequately, preferably by corrugated rubber sheeting. If removal of the appendix is difficult, or unwise in view of the patient's general condition, it may be safely left and drainage alone instituted. Although a less desirable procedure, it does not affect the prognosis materially. I have several times been obliged to leave the appendix, but have not lost a patient by so doing.

The prognosis is, on the whole, quite good, *taking into account the age of the patient* and the poor reaction which is so typical of the class under consideration.

While nearly all my cases fitted more or less closely into the group I have described, there have been two others in which the slow reaction was so exaggerated as to present still another picture altogether. Although the condition is rare, I have thought it worth while to record the salient features.

In both cases there was a history of pain, more or less continuous in the right iliac fossa for several months, associated with constipation and occasional diarrhœa. The onset of the illness was mild, and could not be definitely fixed. On examination there was found a mass in the right iliac fossa, which was hard and fixed, but not very tender, and was to all appearances a neoplasm in the region of the ascending colon. The first was, after investigation, operated upon as such, but an appendix abscess was found. The contents were odourless and composed chiefly of mucus and were probably sterile. In the second case, between first seeing the patient and admitting her to hospital, a period of three weeks elapsed, and in that time the swelling, which had been very marked, almost completely disappeared. In her

case it was found at operation that only a very small quantity of pus remained, and that the abscess had practically resolved spontaneously.

Here again, arrival at the correct diagnosis depends largely on having in mind the possibilities of an extremely chronic inflammatory condition in the appendix. The absence of occult blood in the fæces is strong confirmation of such a state of affairs.

It is quite safe to temporize with these cases, as by postponing operation resolution may occur; but it is wise to remove the appendix at a later date, as it nearly always gives rise to symptoms and may at any time flare up again.

SUMMARY.

1. Consideration is given to anomalous forms of appendicitis in old people.

2. Their similarity to cases of acute obstruction is noted.

3. The symptoms are reconciled with the pathological findings.

4. The treatment is indicated.

A CONTRIBUTION TO THE SURGERY OF THE PITUITARY REGION.

AN ACCOUNT OF FOUR CASES OF 'PITUITARY TUMOUR' TREATED BY RADON SEEDS.

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Owing to the recent death, and autopsy, of the third case of this series, the time has arrived when the four cases can be reported, all operated on during the last six months of the year 1929. They form, I believe, the first series of 'pituitary tumour' cases in which an attempt has been made to improve the condition of the patient by the introduction of radon seeds into the tumour mass. This short contribution to the surgery of the pituitary region deals solely with the technique, etc., of radon-seed introduction, and the results obtained, together with some comments.

In the experience of surgeons interested in the surgery of the pituitary region, it would appear that: (1) 'Pituitary tumours', in their various phases and aspects, are on the increase; (2) Their recognition is more readily made than heretofore; and (3) The ordinary surgical procedures are associated with a very undesirable mortality-rate and also unsatisfactory in their end-results. Consequently any new procedure merits consideration.

The introduction of a radon-seed manufactory at St. Bartholomew's Hospital suggested the possibility of a trial in respect of 'pituitary tumours', and a case soon presented itself that fully justified the attempt (*Case* 1). The result obtained was sufficiently encouraging, and three other cases were treated in the same manner in the same year. The four cases not only demonstrate the feasibility of radon-seed introduction, but also show that some benefit results—for example, the first case so treated was obviously dying when in hospital, and yet lived nine months in comparative comfort; whilst the fourth case, now living in Ireland, has benefited very greatly. It is impossible from this number of cases to claim more than this, and it must be open to question whether the improvement observed in these two cases could not be accounted for by the general decompressive action of the operation. Further experience is needed before this question can be settled.

It is necessary, as a preliminary, to be quite clear as to the justification of any major procedure, whether the ordinary attempt at tumour exposure followed by partial extirpation by sucking or electro-surgery, the introduction of radon seeds, or any other similar measure, and the indications as laid down by Sir Percy Sargent may be taken as a sound guide—for the relief of intolerable headache, and for the saving of the eyesight when the tumour

is progressing in the forward direction and threatening the optic nerves and the optic chiasma.

In all four cases the same method of seed introduction was carried out—a lateral frontal bone-flap (right in three cases, left in one), the mesial margin of the flap falling short of the middle line, thus avoiding the superior longitudinal sinus, and the lower border of the flap passing close above the supra-orbital margin—the flap turned outwards. If the frontal sinus is exposed, the cavity is sterilized and packed with gauze till the termination of the operation.

The dura mater enclosing the frontal lobe is raised from the orbital plate as far back as the posterior border of the anterior fossa, and the dura and brain are retracted with the aid of a special curved metal retractor with light attached—the pattern illustrated was made for me by Messrs. Allen & Hanburys, and it forms, I think, a distinct advance on other illuminating retractors (*Fig.* 37). The wires are enclosed in rubber tubing, and the whole can be sterilized by immersion in lysol, etc. The wires are connected with a small dry-cell battery concealed in a pocket in the operating gown.

FIG. 37.—The illuminating retractor.

The basal region of the anterior clinoid process is verified, and a small incision made in the dura, as near to the base of the process as possible. Through this slit a blunt director is passed, directed backwards, inwards, and downwards, till it strikes the bone at the base of the pituitary fossa. Whether the tumour is of the body itself or of the suprasellar region is estimated beforehand; this will naturally influence one in the direction of the guide.

Previous experimentation on the cadaver showed that a blunt instrument could be thus introduced into the pituitary fossa without material damage to the various important anatomical structures related thereto. The guide passes above and internal to the cavernous sinus and the structures in relation to it, anterior to the front part of the circle of Willis, and with but slight damage to the brain (temporo-sphenoidal pole). Neither the brain of *Case* 2 (in the Museum of St. Bartholomew's Hospital) nor that of *Case* 3 (in the Museum of the West End Hospital for Nervous Diseases) shows other than the slightest degree of cerebral lesion. The second case certainly died shortly after the operation, but the small amount of blood effused at the base of the brain was derived from a vessel of the tumour itself, bursting probably as the result of the general decompression.

Along the track of the director the special seed-introducer is passed, and

the seeds are expelled—spaced as far as circumstances permit, by lengthening or shortening the introducer and by alteration of its angle to the base of the skull. That this spacing can be carried out is proved in all four cases, as seen on the X-ray pictures (*see* especially *Figs.* 38, 39).

Number of Seeds Introduced.—Fourteen seeds in *Case* 1, 9 in *Case* 2, 8 in *Case* 3, and 8 in *Case* 4, each seed representing 1 millicurie radon emanation. The only guide to the number of seeds requisite for the case is the degree of bone destruction, as representing the size of the tumour mass, assisted perhaps by the intensity of the symptoms. The two autopsies showed, however, quite convincingly, that many more seeds were required to include the main mass of the tumour than were actually utilized. This is one of the lessons learnt from the four cases. On the other hand, the surgeon having introduced a certain number of seeds with safety to the patient, naturally hesitates to insert the greater number.

In all four cases the diagnosis was clear, two sellar tumours and two suprasellar, and no attempt was made at the operation either to see the tumour or ascertain its dimensions. It is possible that this method of 'blind insertion' may be criticized, but it should be noted, in defence, that the exposure of the tumour adds materially to the danger of the operation, and that, in any case, it is usually quite impossible to determine the size, shape, and extent of the tumour mass.

Further points in respect of the number of seeds used, their spacing, etc., will be brought out in the description of the four cases.

CASE REPORTS.

Case 1.—Admitted into St. Bartholomew's Hospital under the care of Sir Thomas Horder, suffering from 'blindness'. A blow had been received over the left frontal region twelve weeks previously, and subsequently the patient had suffered from ever-increasing headaches and loss of sight. There was marked proptosis of both eyes.

On Examination.—Without going into unnecessary clinical details, the neurological report was to the effect that the neoplasm was not exactly of the pituitary substance but rather of the base of the skull, invading the base in the downward direction, and upwards into the substance of the base of the brain in the immediate neighbourhood of the pituitary fossa. The X-ray report stated that " the sphenoidal sinus appears clear. The pituitary fossa is not recognized, and the whole of this part of the sphenoid, including both anterior and posterior clinoid processes, has disappeared. The appearances are consistent with a tumour eroding the pituitary fossa and encroaching upon the sphenoid, suggesting malignancy."

Operation (Oct. 10, 1929).—After consultation with Sir Thomas Horder and Dr. Finzi, I determined to see whether radon seeds would do any good. The patient was much worse, very drowsy, with increased bulging of both eyes, incontinence of urine and fæces, and at times quite unconscious. During the operation periodic blood-pressure tracings were taken, to act as a guide as to whether the introduction of the seeds could be completed or not. The following are the records obtained :—

At the beginning of the operation ..	140/90
On the turning back of the bone-flap ..	140/90
On lifting up dura and brain	120/80
On introducing the seeds	130/90
At the termination of the operation ..	190/85

The patient was X-rayed the following day, the report being " fourteen seeds can be identified in the middle line, two-thirds just anterior to the pituitary fossa,

and six lateral, three on either side." The accuracy of the insertion and the spacing was encouraging (*Figs.* 38, 39).

SUBSEQUENT HISTORY.—For two or three days after the operation, the patient was worse and expected to die, but on or about Oct. 20 he improved to a marked

FIGS. 38, 39.—*Case* 1. Lateral and antero-posterior views of the skull. Fourteen seeds situated in the pituitary region. The pituitary fossa and its osseous boundaries are destroyed by growth. The seeds are accurately placed and well spaced.

degree, talked rationally, and the bulging of the eyes was much less obvious. Subsequently his condition improved to such an extent that he was discharged to his home in the Midlands. There further improvement took place: the headache was relieved and the general condition became so much better that he was enabled to go to local football matches, on which game he was an enthusiast. Six months after the operation he fell dead in the street. There was no post-mortem examination.

Comment.—Whether the improvement that took place after the operation was due to the effect of the seeds or the decompression is open to question, but the feasibility of seed introduction was demonstrated, and it was also clear that the seeds could be inserted with fair accuracy and spacing. These facts encouraged me to try again.

Case 2.—A young girl, age 13, was admitted to hospital under the care of Mr. Foster Moore, in September, 1929, suffering from severe headache and loss of vision. The symptoms commenced in May of the same year. The headaches became ever more severe, mainly frontal in position, and progressive eye trouble induced her to seek advice.

On Examination.—It was found that the girl was suffering from marked papillœdema in both eyes, 2 to 3D. This steadily increased and was followed by paralysis of the left sixth nerve. The visual fields showed well-marked bitemporal hemianopia. The X-ray report stated that "the pituitary fossa is definitely

Figs. 40, 41.—*Case* 2. Lateral and antero-posterior views of the skull, showing nine seeds, accurately placed and well spaced.

enlarged for a child of this age, with apparent erosion of the posterior clinoid process—also abnormal shadows above the fossa, the appearances strongly suggestive of a pituitary tumour" (the tumour proved to be a suprasellar adamantinoma).

Operation (Nov. 20).—Nine radon seeds were inserted, without any difficulty, as in the previous case. I felt that the seeds had been inserted into a tumour mass, as definite resistance was encountered by the introducing instrument. The patient left the operating theatre in excellent condition, but during the night respiration, pulse-rate, and blood-pressure all showed a high grade of intracranial pressure, and the patient died.

Autopsy Findings.—In view of the importance of the case, X-rays were taken shortly after death (*Figs.* 40, 41). At the autopsy the brain was removed and hardened, and subsequently X-rayed. A large suprasellar tumour was seen, with all nine seeds buried in the tumour, seven in the ipsolateral half of the tumour and two in the middle line (*Figs.* 42–44).

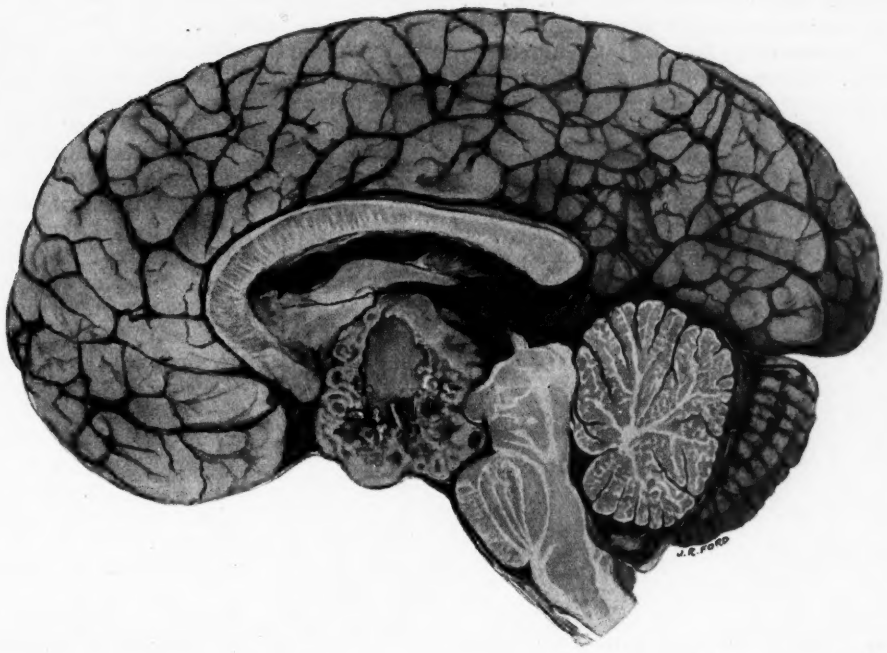
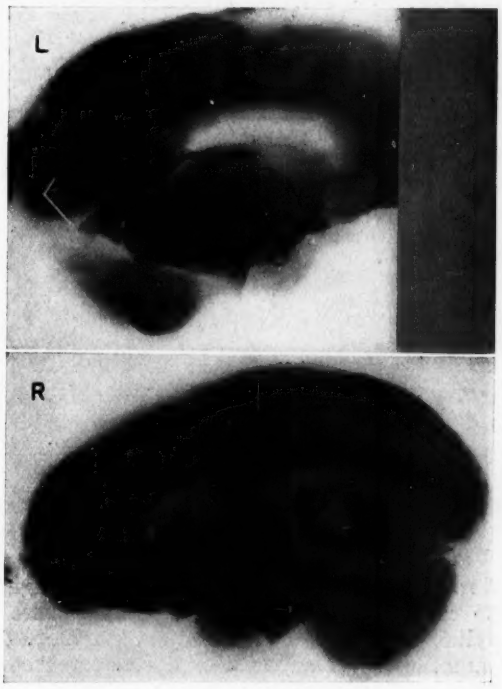


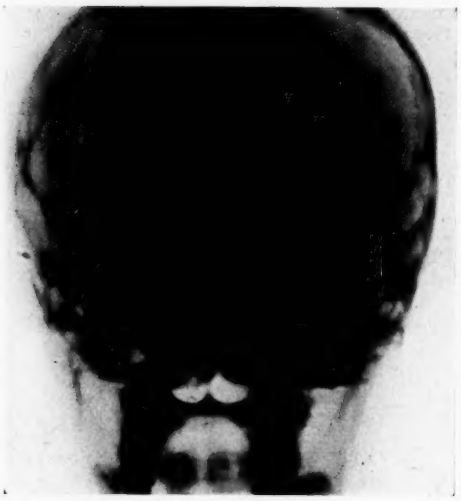
FIG. 42.—*Case* 2. Two seeds are seen in the centre of the tumour in the middle line. (*Drawn from the specimen in the Museum of St. Bartholomew's Hospital.*)

Comment.—This case again demonstrated conclusively that the seeds could be implanted accurately. The cause of death was not quite clear. There was some hæmorrhage at the base of the brain, but the source of this bleeding could not be determined; it looked as if some vessel of the tumour itself had burst as the result of the general decompression.



FIGS. 43, 44.—*Case* 2. Right and left halves of the brain. A wire circle surrounds the tumour mass, and it can be seen that the contralateral half of the tumour (on the side opposite to the bone-flap) contains no seeds, but that all the seeds introduced (eight in number) are situated in the tumour substance on the ipsolateral side. The ventricle is markedly dilated.

Case 3.—A boy, age 8, was admitted to the West End Hospital for Nervous Diseases, under the care of Dr. C. C. Worster-Drought. He had suffered from severe headaches with vomiting for the previous ten months. The sickness came on in the morning, followed by headache, mainly frontal in site, and then screaming attacks. There were intervals in which he appeared to be better, but he became progressively worse. Sometimes he suffered from vertigo, falling always to the right. He was very irritable, emotional, and often hysterical.



FIGS. 45, 46.—*Case* 3. Lateral and antero-posterior view of the skull. Seeds well spaced, but more widely spread than in the previous cases.

ON EXAMINATION.—It was found that he had marked papillœdema in both eyes, equal to 4D. Fields normal except perhaps for a slight narrowing in the temporal fields. Headache more or less constant and vomiting of the cerebral type.

July 12.—The patient was very drowsy all day. The X-ray picture showed enlargement of the pituitary fossa and calcification immediately above the sella, suggestive of the calcification of an adamantinoma of the suprasellar region.

OPERATION (July 17).—Eight radon seeds were inserted, without any difficulty.

July 19.—He seemed much better and stated that he could see better.

July 27.—Improvement maintained. Considerable bulging in the region of the bone-flap. Papillœdema 2D.

Aug. 16.—Much better, little headache, and mentality much improved.

Sept. 10.—Discharged from hospital.

RE-ADMISSION.—The boy was re-admitted on Nov. 30. His progress since discharge could be summarized as follows: First week: not well, headaches. Second week: very well. Third week: severe headaches, vomiting, and screaming attacks.

Subsequently there was some improvement, he smiled at times, and answered questions. Pupils dilated and discs white. Owing to the increasing forward bulging of the bone-flap, the flap was turned down and the bone removed.

The lad remained in hospital during the whole of the year 1930, steadily going downhill. Owing to the progressive bulging at the wound, various efforts were made to reduce this, both by lumbar puncture and intravenous injection of hypertonic saline solution. For example, on Dec. 4, the following was noted after lumbar puncture. Withdrawal of 5 c.c. cerebrospinal fluid—headache relieved; 10 c.c.—flap bulging reduced; 20 c.c.—further reduction; 30 c.c.—severe headache.

Dec. 5.—Injection of 50 c.c. of a 30 per cent hypertonic saline solution was followed immediately by increased bulging of the flap, marked headache, and vomiting.

Dec. 6.—The same measure was repeated with even more severe results, these being at once alleviated by the withdrawal of 40 c.c. of cerebrospinal fluid.

Dec. 15.—Intravenous injection of 50 c.c. of a 15 per cent hypertonic solution. At 45 c.c. there was no change, but at 50 c.c. slight headache and some increased bulging of the flap.

The X-ray picture showed that the eight seeds were arranged as follows: Three in the fossa, three in relation to the anterior clinoid process, and two in relation to the posterior (*Figs.* 45, 46). The lad died early in 1931.

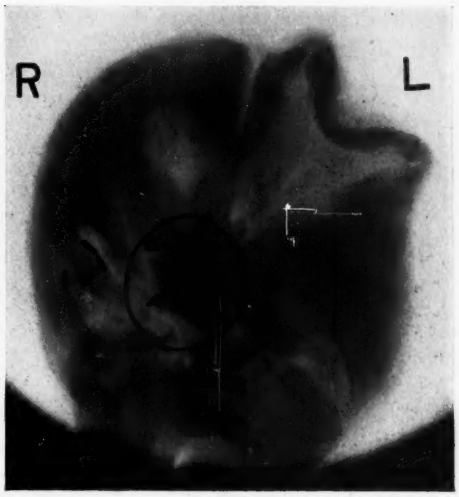


FIG. 47.—*Case* 3. Skiagram of the brain, showing five seeds within the wire of the tumour circle, the great dilatation of the ventricles, more especially in the forward direction on the side operated on, and three seeds apparently extruded into the contralateral ventricle.

AUTOPSY FINDINGS.—At the post-mortem examination a large suprasellar tumour was found, calcified at its base and extending into the ventricular area, the ventricles themselves being markedly dilated, more especially towards the area which had been decompressed.

The brain was X-rayed. Difficulty was experienced in arranging the brain in a natural position; it was very soft, and the forward bulging of the left frontal pole added to the difficulties. The picture showed, however, that five seeds were concealed in the tumour mass, within the wire circle, but that three had wandered laterally and looked to be lying free in the dilated ventricle (*Fig.* 47). The specimen is being hardened and examined further.

Case 4.—A man, age 31, was admitted to the West End Hospital for Nervous Diseases under the care of Dr. Worster-Drought. A year and a half previously, when in Kenya, he was operated on for persistent headache—subtemporal decompression, with at first considerable improvement. Subsequently, the headaches returned; he suffered from giddiness and impaired vision.

ON EXAMINATION.—It was found that the patient had papillœdema in both eyes, 1 to 2D. Visual fields normal. X-ray showed marked sellar enlargement and definite local destruction of bone.

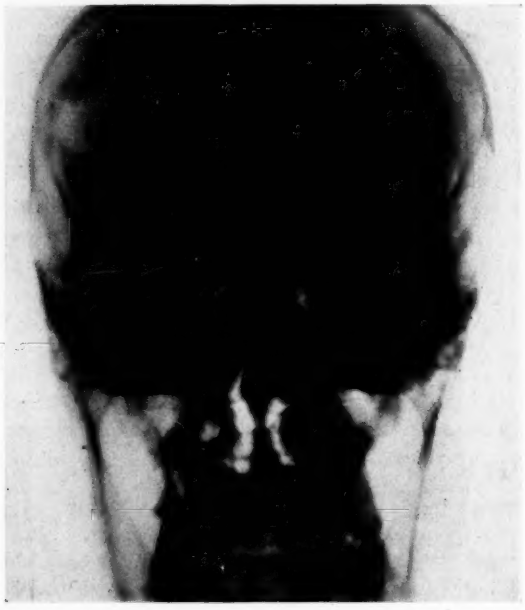
OPERATION (Nov. 11).—Eight seeds were inserted, without difficulty. Subsequent to the operation there was marked improvement in all respects. X rays showed that the seeds were in fair position (*Figs.* 48, 49).

SUBSEQUENT HISTORY.—The after-history of this patient is interesting. In the first place, early in December, he developed marked polydipsia and polyuria. Up to the end of December the intake averaged about eighteen pints during the twenty-four hours, with corresponding output. In all other respects he felt well. He was then discharged.

The present condition of this patient, from a letter received (from Ireland) in December, 1930, more than one year after the operation, runs as follows: "My general health is good, my sleep is only fair. My weight when entering the hospital was 9 st. 2 lb. My weight is now 11 stone. My vision seems to be somewhat better, and I think the glasses have helped them. My thirst and abnormal passing of water has now returned to normal. My gait is very much better but not yet perfect. I am a good deal steadier. I am doing a good deal in the line of exercise and did a good deal of swimming in the summer. I walk on an average

nine miles a day. In every way I feel very much better—hearing, speech, strength, everything I can think of."

The eye-report reads as follows: "Full fields. Nystagmus on looking to the right, also present but less marked on looking to the left. Pupils equal in size, each



FIGS. 48, 49.—*Case* 4. Lateral and antero-posterior view of the skull, showing eight seeds in the pituitary region, well spaced, though two seeds are rather lateral. The decompressed region of the skull (operation in Kenya) is seen in both figures.

reacts to light and convergence. The discs are lacking in sharpness of outline, and there is, I think, slight narrowing of vessels, especially in the left eye—appearance is that seen after papillœdema that has been relieved by decompression."

Comment.—Altogether this case may be regarded as highly satisfactory. In respect of the polydipsia, etc., I have observed this phenomenon on a previous occasion. A patient was admitted to St. Bartholomew's Hospital under my care suffering from a fracture of the middle fossa of the skull. There was bleeding from both ears, and it was clear, from researches carried out by me many years ago, that the fracture passed through the body of the sphenoid. This patient developed polydipsia, etc., within a day or two of admission to hospital, and this continued for the next ten days or so, graduaally returning to the normal and ceasing in about three weeks. It was considered at the time in this case that the pituitary body had been injured, and in *Case* 4 it seemed clear that the introducer or the seeds had brought about a similar change in the pituitary function.

CONCLUSIONS.

From a general résumé of these four cases, it is clear :—

1. That radon seeds can be introduced into and spaced throughout a tumour of the pituitary region.
2. That the neighbouring structures can be avoided.
3. That the results obtained are not discouraging.

TRAUMATIC ARREST OF EPIPHYSIAL GROWTH AT THE LOWER END OF THE TIBIA.

By BRYAN McFARLAND,

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It has always seemed interesting that while a very small proportion of injuries to the lower end of the radius during the period of epiphysial growth are followed by a progressive deformity, the reverse is true of the tibia. After trivial trauma at the ankle a deformity giving rise to serious disability may arise. The proportionate rarity of similar trouble in the wrist is due to the difference in the shape of the joint and in the nature of the injury. There

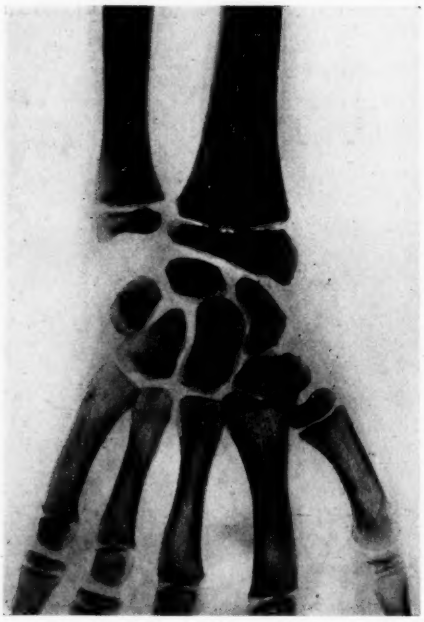

Fig. 50.—The type of fracture which gives rise to deformity similar to that shown in *Fig.* 02.

Fig. 51.—Injury sustained seven years previously. This man did full work as a mechanic.

is no 'mortice' at the wrist-joint, movements are free, and a fall usually displaces the epiphysis and growing cartilage completely with a slice off the diaphysis. Only when the epiphysis is crushed does arrest of growth occur (*Fig.* 50). The deformity which then ensues consists of radial deviation and prominence of the lower end of the ulna. Possibly some of these cases are

classed as Madelung's deformity. There is very little disability. In one case the man earned full wages as a mechanic (*Fig.* 51).

Occasionally the lower tibial epiphysis is bodily displaced (as is usual at the wrist), and it can then be shown that there is no subsequent interference with growth.

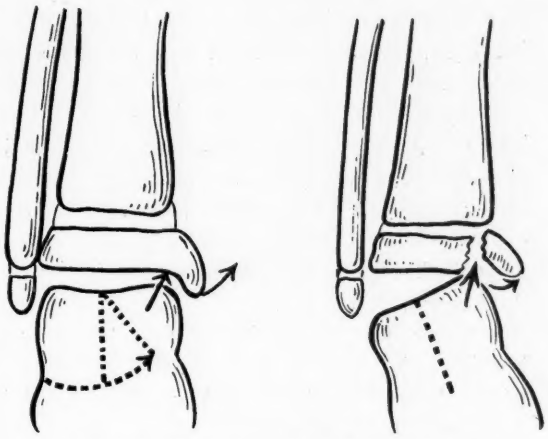

FIG. 52.—Mechanism of epiphysial fracture.

On the other hand, at the ankle the astragalus is firmly held by the tibial and fibular malleoli. Lateral movement is not free, and if it is forced, an undue strain is put upon the malleoli. Where children are concerned, this strain falls on epiphyses, and it is found that the injury preceding tibial arrest almost always consists of an inward twist of the foot. The astragalus is forcibly adducted, the inner edge of the upper surface impinges on the inner half of the epiphysial plate of the tibia and applies a crushing force at this point (*Fig.* 52). At the same time the internal malleolus is forced upwards and inwards and the epiphysis breaks at the base of the malleolus.

FIG. 53.—Injury one year previously.

It was interesting to hear from Willis Campbell that this epiphysial fracture is, in his opinion, rare in the United States. In this country cases occur fairly frequently, and there are 23 cases in my series (two years—about 3000 fractures). For some time this discrepancy seemed inexplicable, but the solution probably lies in the causative agent. This is in nearly all cases a railing composed of regularly spaced vertical iron rods used to enclose gardens and parks; the children are addicted to climbing over them and slip with one foot jammed between the two rods. Such railings abound in England, and are apparently extremely scarce in America. The constancy of the history is well illustrated by the following incident:—

I was asked to see a boy as an out-patient who had hurt his leg the previous day, and on my way was shown the skiagram (*Fig.* 53); this showed that he had injured his epiphysis about a year ago. I informed the house surgeon that in all probability his recent injury could not be responsible for this X ray. Before the patient was examined his older brother was asked if about a year ago he was climbing some railings and slipped with his foot caught fast. The brother immediately remembered the incident

as he had had to free the boy and carry him home. The boy had limped for a week or so, had then apparently recovered, and the whole thing had been dismissed as a sprain.

This is the typical history. The disability is transient and slight at the time. After a year a deformity may be noticed and is seen gradually to increase, the attention of the parent being drawn to the 'going out' at the ankle and the uneven wearing away of the boots.

It will be seen from the skiagrams that, following the crushing of the inner half of the epiphysial disc of the tibia, this half only ceases to grow, presumably owing to the death of cartilage cells, either from being crushed or from hæmorrhage into them. The outer half and the fibula continue to grow at the normal rate, and the resulting deformity is what would be expected. The ankle-joint turns inwards just as surely as a door swings on its hinges.

Thus a varus deformity of the ankle develops owing to a low outward bowing of the tibia and fibula. The external malleolus may be as much as $2\frac{1}{2}$ in. below the level of the internal. The astragalus goes with the malleoli, and its vertical deviation is incompletely corrected at the mid-tarsal and talo-calcaneal joints.

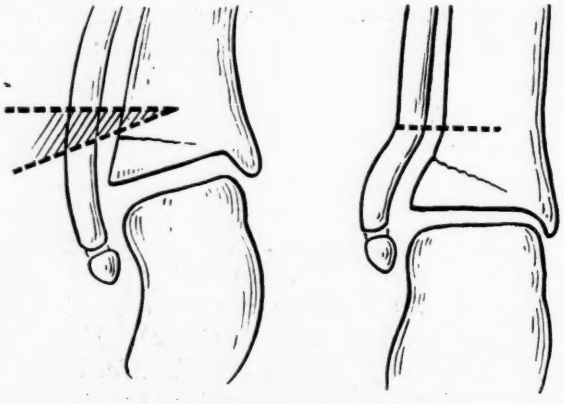

FIG. 54.—Diagram showing the method of operative correction of the deformity.

The treatment of the acquired deformity is simple and efficacious. Through an external incision the fibula is exposed just above the ankle-joint. A wedge consisting of fibula and three-quarters of the thickness of the tibia is removed, as in *Fig.* 54. The line of this passes through the interosseous ligament where the two bones are practically in contact. The upper surface of the wedge should be horizontal, the lower parallel with the lower tibial articular surface.

Unless the patients are importunate, operation should not be done in cases under 14 years of age, and it is best to wait until 18 to 20 years, when epiphysial growth has ceased. One case under 14 was corrected, and in order to minimize the shortening of the leg a piece was taken from the fibula on the outer side, the tibia was divided from the inner side, deformity was corrected, and the piece of bone was put into the wedge-shaped gap in the tibia.

Attempts have been made, as one would expect, to prevent the occurrence or progression of the deformity. These have not been definitely successful.

The following skiagrams (*Figs.* 55–64) are a selection from a large number of cases illustrating the constancy both of the type of injury and of its resulting progressive deformity.

FIG. 55.—Twisted foot in railings a month previous to photograph.

FIG. 56.—Same case as *Fig.* 55, seven months after accident.

FIG. 57.—Same case as *Fig.* 55, two years after accident.

FIG. 58.—At the age of 10 this patient was climbing railings when he fell, and one foot stuck. Nine years after accident.

FIG. 59.—Same case as *Fig.* 58. Corrected January, 1929. Photograph five months later.

FIG. 60.—A case successfully treated by osteotomy. Fifteen years after accident.

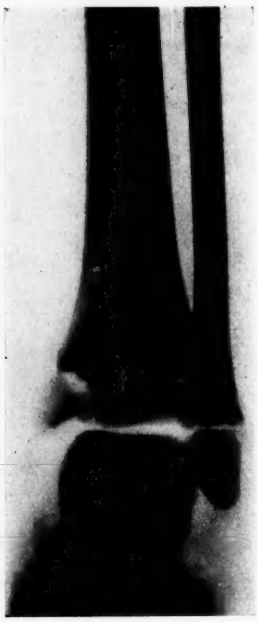

FIG. 61.—The patient twisted his foot in a grid. Photograph a few days after accident.

FIG. 62.

FIG. 63.

FIG. 64.

FIGS. 62–64.—Same case as *Fig.* 61. *Fig.* 62 shows the condition two years after accident. *Fig.* 63, four years after accident; parents anxious for correction; osteotomy in June, 1928. *Fig.* 64, one year after correction by cuneiform osteotomy of fibula and insertion of the piece of bone into the gap of a linear osteotomy of the tibia.

CARCINOMA OF THE THYROID GLAND.

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(*Being an Arris and Gale Lecture delivered at the Royal College of Surgeons of England on February 4, 1931.*)

More than ordinary interest attaches to carcinoma of the thyroid gland because it has been stated that it has features which are not known to occur in malignant disease of any other organ. Some of these, if true, would destroy the basis on which our ideas of malignancy are founded. It has been asserted that a benign tumour of the thyroid, and even a normal gland, may give rise to metastases. This belief is associated with names distinguished in surgery and pathology such as Lucke, Cohnheim, von Eiselsberg, Kraske, and others. It is now generally agreed that a secondary growth may reproduce some of the morphological characters of the primary growth, but it has been stated that the thyroid malignancies do more than this in that the metastases may be indistinguishable from the normal gland tissue. A malignant growth should have no physiological activity, yet von Eiselsberg has shown that a metastasis from a malignant thyroid tumour can function to an extent adequate for the body's needs after extirpation of the parent gland, and thyroid carcinoma tissue has been proved to have functional activity by the tadpole test.

The case reports upon which some of the above assertions were founded have been critically examined by Bérard and Dunet,[1] Joll,[2] Graham,[3] and others, and reviewed in the light of their own experiences. It is clear from their results, and from our own investigations, that some of the apparently anomalous features of the pathology of the thyroid are to be attributed to the fact that the character of the cells and their arrangement vary so greatly in growths that are benign. The extent of this variation may be such that it becomes difficult to determine when the activities of a growth have crossed the line dividing innocency from malignancy. For much of the recent work devoted to this aspect of the subject we are indebted to Allen Graham,[3] at Cleveland; Simpson,[4] at Ann Arbor; Aschoff,[5] Wegelin,[6, 7] and Bérard[8], on the Continent; Scott Williamson and Innes Pearse,[9] in London; and Tebbutt,[10] in Sydney.

The purpose of this paper is not to cover the whole field of carcinoma of the thyroid, but rather to present those observations which have been made on the clinical and pathological material occurring in my work. This material has been gathered while working in the Surgical Professorial Unit of St. Bartholomew's Hospital, and before that in Australia. It is inevitable in clinical investigation that gaps in our inquiries and observations on individual patients should occur, and therefore finality is not claimed for the

views presented. The diversity of the histological picture adds a peculiar difficulty to the investigation of the pathology of the thyroid gland, and I am indebted to my colleagues of the pathological department for their assistance.

Classification.—The classification of carcinoma of the thyroid varies in every country, and with every author I have read. Many classifications are long and confusing. The reason for this is that comparatively few workers have directly concerned themselves with this problem. For the most part each has worked more or less alone and has been intent upon the material of his own clinic, and each has evolved a classification which seemed best to meet his needs. Nevertheless uniformity is beginning to appear.

The classification I have adopted is similar to that published by Allen Graham.[3] It is adequate, and there is neither necessity nor reason ofr the more complicated subdivisions adopted by many. This classification divides the carcinomata into: (1) Scirrhus (or carcinoma simplex); (2) Papilliferous adenocarcinoma; (3) Malignant adenoma.

Objections can be raised against this classification in that the second and third groups tend to overlap. This overlap occurs because, whatever the stimulus to cell multiplication, or the type of carcinoma induced, thyroid epithelium retains to some extent a capacity to produce any of its morphological characteristics—that is, the cells may be arranged in anastomosing columns, or in follicles, or the structure may become papilliferous. Notwithstanding this, each type has features sufficiently outstanding to place it in its class.

Criteria of Malignancy.—The question will continually arise: On what criteria is the diagnosis of malignancy based? If we are satisfied with the descriptions usual in surgical text-books—which almost invariably refer to the later stages of the disease—no difficulty arises, but by that time the information is useless to the patient, and practically devoid of interest to the investigator. If we are to attempt to recognize cases of malignancy at an early stage—and surely this is our most urgent need—difficulty is met at once. The chief reason for this is that, apart from scirrhus (carcinoma simplex), carcinoma rarely arises in a normal gland; it almost always occurs in a gland which is already pathological and in which the epithelium, although benign, may show extremely diverse types of structure (*see Figs.* 90, 91), and the change from a benign to a malignant nature may take place with scarcely any change in the character or arrangement of the cells. When the morphological appearances may be so similar in a malignant and a non-malignant tumour, the criteria of malignancy must sometimes rest, not on the character or arrangement of the epithelial cells, but pathologically on such evidence as invasion of the capsule and invasion of vessels, and clinically on local recurrence or metastases. In both papilliferous adenocarcinoma and malignant adenoma the ultimate criteria are the same, but the difference in the characteristics of the two types is such that each presents its own difficulty in drawing the line between innocency and malignancy, and in each case epithelial activity has been present long before a pathologist would give a definite opinion that malignant transformation had occurred. It may be right for a pathologist to be conservative and refuse to accept a diagnosis until the

proof is beyond doubt, but the welfare of the patient demands that the biological side of the question should be given adequate consideration.

Origin of Thyroid Carcinomata.—

1. *Scirrhus.*—Scirrhus (carcinoma simplex) originates in the same manner as scirrhus of the breast. The epithelium breaks through the basement membrane of the alveoli.

2. *Papilliferous Adenocarcinoma.*—From our own observations we know that an innocent papilloma (or one not proved to be malignant) has originated in an adenoma. In some of our cases of papilliferous adenocarcinoma I have not been able to obtain a history of a preceding tumour. Also, I have found papilliferous growth, apart altogether from tumour formation, almost as extensive and active as that found in a tumour (*see Fig.* 68). Again, as will be seen later, in our aberrant thyroids papilliferous change had been present throughout all the nodules and the parent gland. Therefore I believe that papilliferous growth can occur apart from adenoma. Wegelin states: "It is certain that the development of the papilloma follows quite independently of the earlier presence of an endemic goitre. The tumours investigated in the Berne Institute are derived for the most part not from the indigenous inhabitants, but from foreigners who have come from goitre-free regions." [6] (Wegelin employs the term 'papilloma', but states that six of his seven cases were malignant; as used by him, therefore, the term practically corresponds to papilliferous adenocarcinoma and not to the benign neoplasm.)

3. *Malignant Adenoma.*—Malignant adenoma invariably originates in a pre-existing adenoma, and in most published records this type constitutes from 85 to 90 per cent of the cases of carcinoma of the thyroid.

Incidence of the Types of Carcinoma.—Scirrhus is comparatively rare. As the percentage of cases of carcinoma arising in a pre-existing adenoma is so high, it will be obvious that the incidence of carcinoma should be higher in those regions where the incidence of endemic goitre with the formation of adenomata is high. Bérard and Dunet[8] conclude that in goitre areas from 2·5 to 4 per cent of all malignant tumours affect the thyroid gland, whereas in goitre-free areas the percentage is only 0·4 to 0·5 per cent. Wegelin[7] states that "these figures speak with no uncertain voice. Where goitre is endemic, malignant tumours of the thyroid occur in greater numbers". In non-goitrous regions I believe that the proportion of papilliferous adenocarcinoma to malignant adenoma is higher, and for that reason the number of cases of papilliferous adenocarcinoma compared with that of malignant adenoma is decidedly higher in my series than in one from a centre of endemic goitre.

Wegelin[6] writes the section on thyroid disease in the *Handbook of Special Pathology* by Henke and Lubarsch. He has an immense amount of material, and he publishes each of his cases of papilloma in detail. There are only seven of these, while it is obvious that his total number of cases of carcinoma is very high. Although, as already stated, he calls these 'papillomata', he shows that six of these have undergone malignant change, and the seventh is a toxic goitre. In our series there have been 9 cases of adenopapilliferous carcinoma in a total of 38 cases.

After excluding doubtful cases, there remain in our series 38 cases, in 32 of which the histories and pathological material are available for study.

This number has occurred in 2127 operations for all types of goitre—that is, 1·78 per cent—but if toxic goitre is excluded it has occurred in 937 patients, being 4 per cent. The latter figure cannot be taken as the average percentage of malignant to non-malignant tumours, because we have been prevented by limitation of beds from admitting any but the more serious cases, or those in which a serious condition has been suspected.

SCIRRHOUS CARCINOMA (CARCINOMA SIMPLEX).

I do not propose to discuss this type at length. As already stated, it is rare in comparison with the other two types. It does not differ from a similar

FIG. 65.—Scirrhous carcinoma (carcinoma simplex).

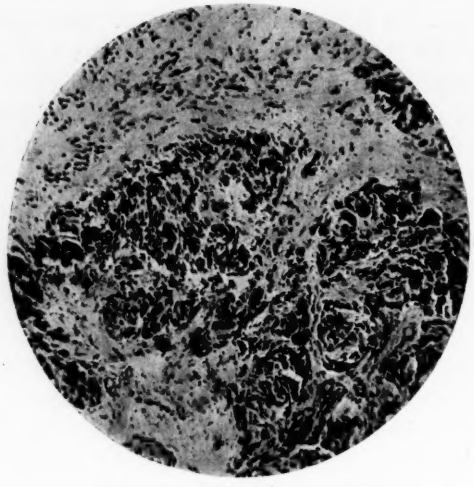

FIG. 66.—Scirrhous carcinoma (carcinoma simplex).

type of tumour elsewhere in the body. The tumour is comparatively small, and very hard. It invades locally, and disseminates to adjacent lymph-nodes. The histopathology is shown in *Figs.* 65, 66.

PAPILLIFEROUS ADENOCARCINOMA.

In our series this type constitutes 23·6 per cent of the total number.

The distinctive feature of this variety of carcinoma is the papilliferous tendency. Papilliferous structure in the thyroid gland is not confined to malignant growths. It occurs in adolescent goitre, toxic goitre, and benign papilloma, as well as in papilliferous adenocarcinoma, and it is almost always present in lateral aberrant thyroids by the time they are recognized clinically. Marine and Lenhart[11] have readily produced goitre in brook trout, and, in some of these, tumours occur of 'arborescent papillomatous' type. It is scarcely likely that this type of epithelial hyperplasia is of different significance in each of these conditions. It rather suggests that stimulation, whether physiological or pathological, elicits this type of response because of the inherent properties of thyroid epithelium.

The papilliferous response of thyroid epithelium in (1) toxic goitre, (2) benign papilloma, (3) papilliferous adenocarcinoma, (4) aberrant thyroid gland tissue, is illustrated by *Figs.* 67–89.

1. TOXIC GOITRE.

Fig. 67 shows a condition which is frequently seen in toxic goitre. *Fig.* 68 is also from a patient suffering from toxic goitre. The extent of

FIG. 67.—Papilliferous formation as frequently seen in toxic goitre.

FIG. 68.—Papilliferous formation in toxic goitre. The extent of the condition in this case is unusual.

the papilliferous formation is altogether exceptional for this condition. This patient—a young woman—had been ill for twelve months, and after four months in bed was becoming progressively worse.

2. PAPILLOMA (BENIGN).

This tumour is classed among the benign neoplasms. In my last 1044 operations in which every specimen removed has been examined microscopically I have found only two cases of simple papilloma, and I cannot be sure that one of these is innocent. The condition, therefore, is rare. In one of these (*Fig.* 69) transformation from vesicles containing colloid to papilliferous structure can be seen taking place along a wedge-shaped front. It is obviously an active process. If an area next to the capsule is examined, no invasion is found (*Fig.* 70). This must be regarded as benign, but active papilliferous change is taking place, and I do not know where (or whether) that activity will stop. Wegelin states that "one cannot say with certainty where the normal tissue ends and the tumour tissue begins."[6] In the next patient the condition had advanced a stage further (*Fig.* 71). We see a similar condition throughout the nodule, but examination immediately beneath the capsule shows great irregularity in the size and shape of the cells, and also what I believe to be invasion of the capsule (*Fig.* 72).

These two points, irregularity of shape and size of cells, and invasion of capsule, are capable of two explanations. The inequality of the cells may be due to degeneration, and, instead of the papilliferous growth invading the capsule, the fibrous tissue may be invading and strangling the growth, but the nuclei of the cells stain so deeply that I think they are not degenerating, and probably the growth is invading the capsule. These two tumours are classed

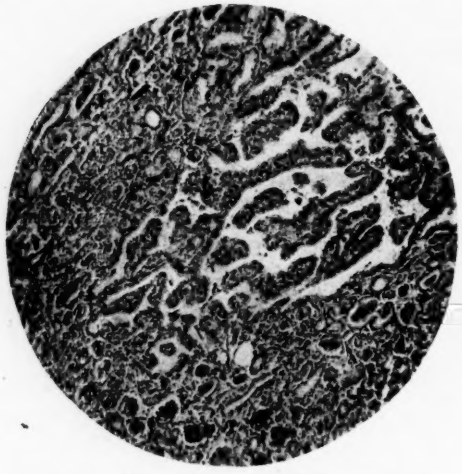

FIG. 69.—A wedge of papilliferous tissue is seen surrounded by vesicles containing colloid.

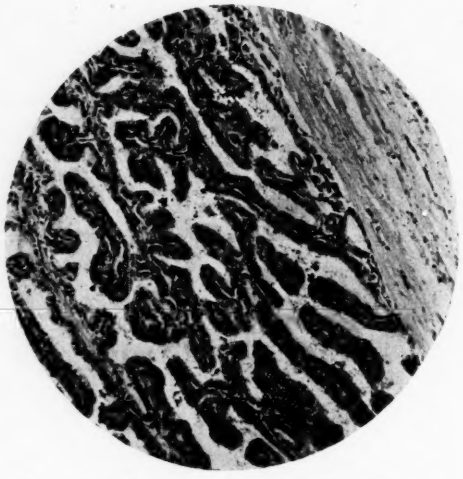

FIG. 70.—The same case as *Fig.* 69. No invasion of the capsule is found.

FIG. 71.—Papilliferous change throughout the nodule.

FIG. 72.—The same case as *Fig.* 71. The epithelium has become more embryonic in type, and invasion of the capsule is occurring.

as innocent papillomata, but the activity of the second case is decidedly suspicious. (Since this article was written this patient has been seen again in the routine 'follow-up' examination. Local recurrence has taken place, confirming our suspicions.) It will be realized how difficult it is to determine where innocency stops and malignancy begins.

3. PAPILLIFEROUS ADENOCARCINOMA.

In the following illustrative cases increasing degrees of activity are shown.

Fig. 73 shows a stage more advanced than *Fig.* 72. In this patient a nodule was present in the isthmus of the gland. This was causing discomfort altogether out of proportion to the size of the nodule. The nodule was removed rather under protest, but patient and doctor were both insistent about the reality of the discomfort. Microscopical examination shows a luxuriant growth of papilliferous nature, and in many places the simple benign appearance has been lost, in that the cells covering the papillæ form multiple layers and the epithelium is growing without restraint. This had passed beyond the stage when it could be classed as a benign neoplasm.

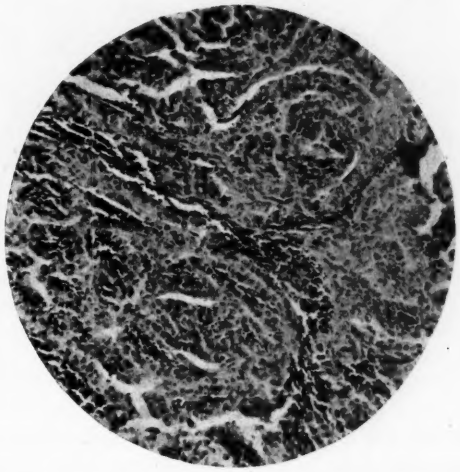

FIG. 73.—Early papilliferous adenocarcinoma.

Next in the series is a tumour in a woman of 64. In this patient growth was more rapid—it had only been noticed eight months previously—and the increase in size had been obvious. The tumour had become fixed to the adjacent structures, and neighbouring lymph-nodes were enlarged. *Fig.* 74 shows papilliferous growth throughout the tumour, but sections taken from the surface of the growth where it was attached to the overlying structures show that it had penetrated the capsule and invaded muscles (*Fig.* 75).

FIG. 74.—Papilliferous growth throughout the greater part of the tumour.

FIG. 75.—From the same patient as *Fig.* 74. Growth has invaded muscle bundles overlying the tumour.

In the third patient in this series the same papilliferous change in the tumour is shown (*Fig.* 76). Malignancy was not suspected before opera-

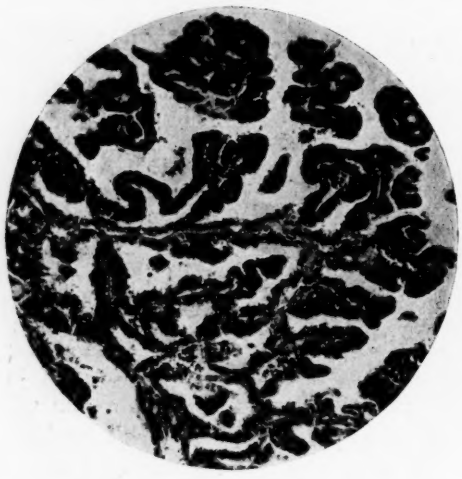

FIG. 76.—Papilliferous change in the third patient.

FIG. 77.—From the same patient as *Fig.* 76. The growth has become more embryonic in character.

tion, and recurrence occurred. On examining the material removed at the second operation, it was found that the greater part of the mass was still papilliferous, but growth had become much more active, and areas were found where dedifferentiation or anaplasia had occurred (*Fig.* 77). The cells were invading the capsule and lymph spaces (*Fig.* 78).

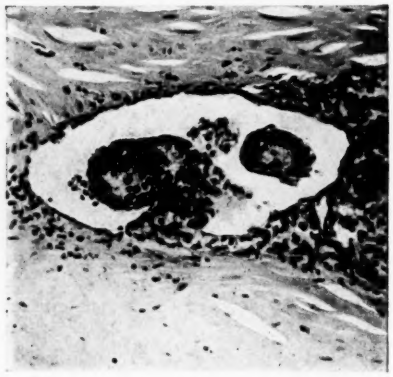

FIG. 78.—From the same patient as *Figs.* 76 and 77. Carcinoma cells in a lymph space.

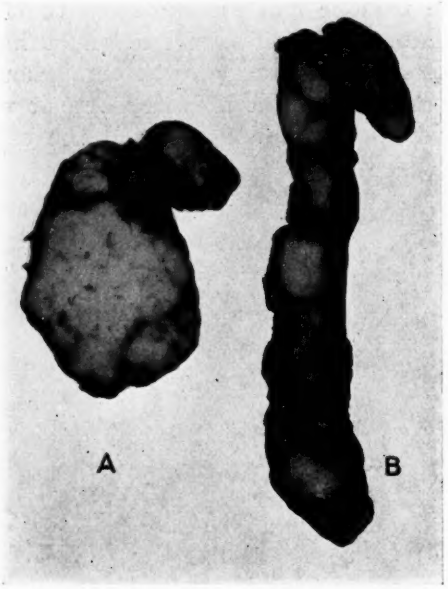

FIG. 79.—Papilliferous adenocarcinoma. A, The thyroid gland; B, Infected cervical lymph-nodes.

In the next example of this type of carcinoma, the thyroid gland and lymph-nodes throughout the neck were extensively involved. *Fig.* 79 shows

the original tumour and a chain of infected cervical lymph-nodes removed at the first operation. *Fig.* 80 shows the microscopical appearance of the original tumour. Section of one of the lymph-nodes shows papilliferous growth replacing the original structure with the exception of a little lymphoid tissue which remains under the capsule (*Fig.* 81).

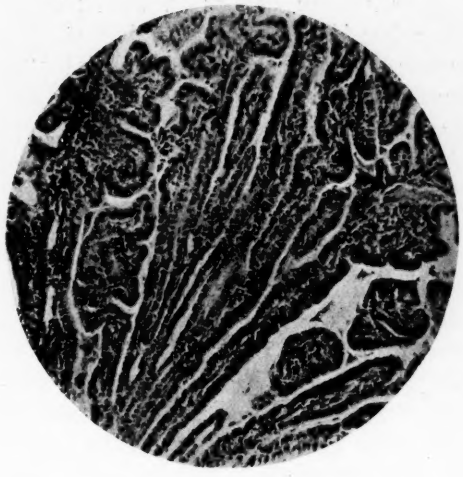

FIG. 80.—The microscopical appearance of the tumour shown in *Fig.* 79, A.

FIG. 81.—The microscopical appearance of one of the lymph-nodes (*Fig.* 79, B).

Discussion of Papilliferous Adenocarcinoma.—

1. It would appear from a study of these illustrations that varying degrees of papilliferous change may be induced in thyroid epithelium.

2. In puberty goitre and in toxic goitre this is probably a response to stimulation, the type of stimulation differing in degree rather than in kind, and in both types it is probably compensatory in nature and occurs because under special stresses the function of the gland is not adequate to the demands made upon it.

3. In both puberty goitre and in toxic goitre the papilliferous condition disappears if gland function becomes normal. The cause is, to some extent, known and under control.

4. An examination of *Figs.* 68–81 shows that a progressive degree of epithelial activity can be traced from the alleged simple papilloma to an invading and disseminating growth. In these conditions the cause is not known, but the histopathological appearance is, in many instances, so similar to that in which the cause is to some extent known (compare *Fig.* 68 with *Figs.* 70 and 74), that we cannot help asking ourselves whether the epithelial hyperplasia forming a papilliferous neoplasm may not result from disordered physiological stimulation of a normal gland, or possibly normal stimulation directed on to a gland—or some portion of a gland—which is subefficient through maldevelopment.

5. These considerations give some reasons for the hypothesis that the epithelial response which appears to us to result in one instance in an

innocent condition may be akin to that which in another results in a malignant condition, the epithelial hyperplasia being an expression of an inherent capacity of the thyroid epithelium, and its activity being aroused by stimulation which may be normal or abnormal.

6. The work of Marine and Lenhart on brook trcut, which has already been mentioned, is interesting in this respect. For many years it had been known that enlargement of the thyroid gland often occurs in brook trout when overcrowding takes place. These workers[11] made exhaustive studies of this condition. The goitre is conspicuous. It consists of epithelial cells of thyroid origin which spread widely but locally. The cells infiltrate and replace muscle bundles and bone in the floor of the mouth and pharynx, and may extend on to the gills. Because of the actual invasion of muscle and bone, the condition was long believed to be adenocarcinoma. In fish the thyroid gland is not encapsulated, and therefore, when hyperplasia occurs as the result of stimulation, the spread of the cells is not limited. When the cause of the hyperplasia is removed, the goitre disappears. Marine and Lenhart decide that, although local invasion occurs, this is not carcinomatous, but a hyperplasia simulating carcinoma because of the peculiar characters of the thyroid gland in fish. Nevertheless in these goitres a true tumour sometimes occurs—papillomatous in type—and this tumour does not disappear with the improvement in hygiene. The cause of this tumour in trout would seem to have something in common with that of the hyperplasia in human beings, and it will be of interest to refer to it again after consideration of lateral aberrant thyroids.

7. Lateral aberrant thyroids. A study of the behaviour of this condition (of which we have had four cases—to be described immediately) gives some suggestion that the response of thyroid epithelium—right up to malignancy—represents an interaction between stimulation and the responsive capacity of the cells, the gland efficiency in the case of lateral aberrant thyroids being limited by imperfect development. It is possible that the information gained from these structures, taken together with the experiments of Marine and Lenhart on trout—admitting that the hyperplasia in brook trout stops short of carcinoma — may help a little way towards an understanding of the evolution of papilliferous adenocarcinoma.

4. LATERAL ABERRANT THYROIDS.

Many cases of this condition have now been reported. Billings and Paul[12] in 1925 reported a series of 35 cases published since 1857, including one of their own. Tebbutt and Woodhill[13] in 1927 reported 5 cases and discussed the literature, together with the clinical condition and pathology of their own cases. In 1928 L. van den Wildenberg[14] reported a case of his own together with 28 cases collected from the literature. In the same year the workers in the Lahey Clinic[15] had collected records of 45 cases, including 4 of their own. In the published cases there have been one or more tumours, usually in the posterior triangle, sometimes situated just above the clavicle, sometimes high in the neck.

We have had four cases of this condition. In each, multiple tumours

have been present, and these have been quite distinct from the thyroid gland, although in all our cases the thyroid gland itself has been enlarged.

Origin of Lateral Aberrant Thyroids. — Whence came these nodules embryologically, and what are they pathologically? At first, because of the lymphoid tissue under the capsule, the colloid vesicles, and the papilliferous growth, it was thought that they were deposits of thyroid carcinoma in lymph glands; but it would seem that these masses are aberrant thyroid tissue, or derive from the pharyngeal entoderm. Morphologically these cystic papilliferous aberrant thyroids are very similar to papilliferous adenocarcinoma occurring in the thyroid.

Writers in Europe and America refer to lateral aberrant thyroids as probably deriving from the ultimo-branchial body—the remnant of the fifth pharyngeal pouch. This belief is not altogether based on demonstrable facts. Langhans had this opinion, and thought that papilliferous adenocarcinoma occurring in the thyroid gland had the same origin. L. van den Wildenberg[14] and the workers in the Lahey Clinic[15] regard this as a strong probability. Wegelin discusses the question at length in the *Handbook on Special Pathology* by Henke and Lubarsch, and in another article in the *Cancer Review*[7] he states: "It appears in most cases to be derived from isolated branchiogenetic rests, especially when it occupies the lateral cervical region." Kingsbury[16] and Badertscher[17] have shown that the ultimo-branchial body becomes fused with the developing thyroid gland in the pig. Grosser[18] (Keibel and Mall) considers it probable that in the migration of cells from the posterior portion of the pharynx to the developing thyroid complete fusion has failed to take place and rests remain. Even though fusion takes place, development may remain imperfect. Williamson and Pearse[9] state that from their view of the development of the thyroid, they expect to find in certain imperfectly developed human glands states corresponding to the more primitive types found in some lower animals. In these conditions, they would expect abnormality of function, and a consequent tendency to malignancy, for they look for one of the predisposing causes to all neoplasia in anatomical and functional abnormalities. The association of lymphoid tissue containing Hassall's corpuscles with this type of thyroid growth is also explained by them on an anatomical basis, for these authors find evidence in the thyroid apparatus of two distinct and separate lymph systems. One of these—the intrinsic thyrothymic lymph system—links the thyroid directly with the thymus, so that the thyroid and the thymus (including the nodes of the thymus scattered in the neck) come to be regarded by them as functionally one organ. Following this theory, they interpret this type of carcinoma as a growth confined, until its later stages, within the lymphatics of the *organ of its origin*, and they believe that this accounts for its anomalous nature, its slow growth, absence of distant metastases, and widespread growth in the mediastinum.

The various theories have been discussed by Professor Woollard,[19] and he has shown that this aberrant tissue need not necessarily derive from the ultimo-branchial body; the potency of the pharyngeal entoderm is such, and the developmental processes are so complicated in that small area, that colloid-producing epithelium attached to or surrounding lymphoid tissue

which may contain Hassall's corpuscles, may be displaced by developing nerves, muscles, or vessels, and lodge in the lateral aspects of the neck.

For my purpose it is not necessary to decide on the actual origin of the entodermal rudiments. The solution of this must remain in the hands of the embryologists, but it is an important question whether these laterally placed rudiments are imperfectly developed, whether tissue of similar origin is incorporated in the thyroid gland, and whether the imperfect development of this tissue leaves it susceptible to malignant degeneration. We are therefore keenly interested in the pathological histology, for it would seem that these misplaced bits of tissue, whether in the lateral triangle of the neck or in the thyroid gland itself, foredoom the host to carcinoma from before the day of birth.

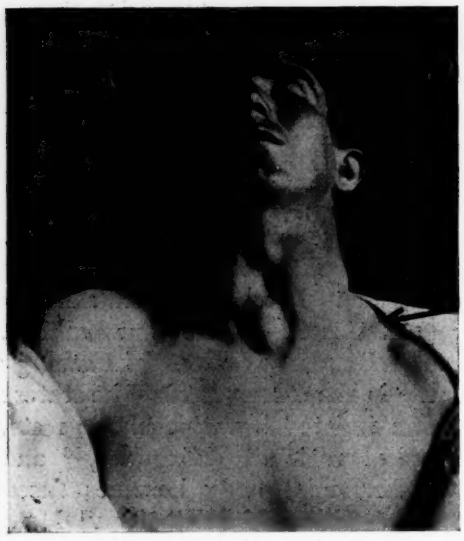

FIG. 82.—*Case* 1. Lateral aberrant thyroids. The tumour situated in the left posterior triangle where the neck joins the shoulder was very conspicuous.

Clinical History and Pathological Histology of our Four Patients.—

Case 1.—The patient was a young man upon whom I operated in Australia some years before the war. It was very early in my surgical life, and I fear I was not then as interested in keeping detailed records as I should have been. I had the patient photographed before excising the lateral masses and a large part of the gland. The masses were cystic and papilliferous, and the gland was of the same nature. *Fig.* 82 shows the nodules in the posterior triangle of the neck on each side.

Case 2.—The patient was 46 years old when I saw her, and she gave a history of at least eight years, so that the condition dated from her early thirties. Masses were present high in the neck behind the angle of the jaw, as well as in the posterior triangles. *Fig.* 83 shows a section from a nodule removed. Paralysis of a recurrent laryngeal nerve was present when this patient was first seen by me.

FIG. 83.—*Case* 2. Section from a lateral aberrant thyroid.

Case 3.—The patient (*Fig.* 84) was 24 when admitted to St. Bartholomew's Hospital, and the condition had been present for seven years, so that it had begun before the age of 17. Several of these nodules were removed. A complete section of one of them is drawn in *Fig.* 85. Lymphoid tissue is present under the capsule. The histology is shown in *Fig.* 86.

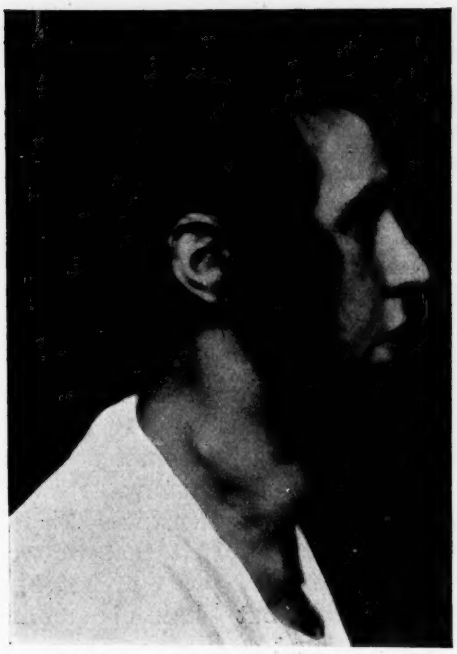

FIG. 84.—*Case* 3. Lateral aberrant thyroid. Thyroid gland also enlarged.

FIG. 85.—*Case* 3. Lateral aberrant nodule. In the stained section lymphoid tissue is clearly seen under the capsule. (× 2.)

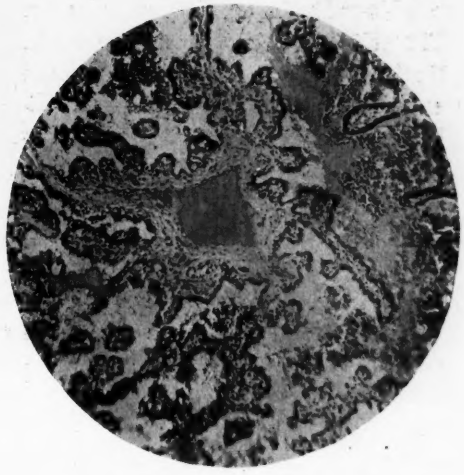

FIG. 86.—*Case* 3. Microphotograph of section taken from the centre of the nodule shown in *Fig.* 85.

FIG. 87.—*Case* 4. Lateral aberrant thyroids. The thyroid gland was also enlarged.

Case 4.—The patient (*Fig.* 87) was 16 when admitted to St. Bartholomew's Hospital, and the condition was known to be present at the age of 12.

In each of these four the condition would appear to be identical. There were nodules of tissue situated in the posterior and anterior triangles of the neck, encapsulated, having no connection, but associated with an enlargement of the thyroid gland. Microscopically the nodules consist of papilliferous growth into cystic spaces, some of which contain colloid, and immediately under the capsule the remains of lymphoid tissue. These tumours are usually classed as papillary cystadenoma with a tendency to undergo malignant transformation.

Taking our four patients: The first was operated upon about eighteen years ago in Australia. I have been unable to trace him since the beginning of the war. The second patient had, when I saw her, involvement of a recurrent laryngeal nerve with paralysis of a vocal cord, implying that the growth was infiltrating. She ultimately died of intestinal obstruction following malignant disease in the pelvis. The third we were able to keep in touch with for three years. She married and had two children, but she regarded our interest in her as far too pathological, and all our efforts to find her subsequently have proved unavailing. The fourth patient was only 16, and the tumours had already been present for at least four years. One nodule was removed for diagnosis. The patient was friendly and interested, and we felt that we were going to be able to keep her under observation indefinitely, but within nine months of the time we first saw her, symptoms of pressure on the trachea began. These increased so rapidly, and were so urgent, that operation became necessary. Five nodules were removed (*Figs.* 88, 89)—making six with the one previously excised—and also the greater part of the thyroid. Relief followed, but pressure rapidly recurred. Deep X-ray therapy failed to give relief, and the patient died.

FIG. 88.—*Case* 4. Lateral aberrant thyroids (with the exception of the largest nodule, which is part of the thyroid gland).

The Bearing of Lateral Aberrant Thyroids on the Question of Adeno-papilliferous Carcinoma.—The embryology is not settled, but it is believed that these bodies originate from the pharyngeal entoderm, and that they have failed to join the median thyroid gland, and therefore to develop perfectly.

All writers agree that cystic papilliferous change takes place in lateral aberrant thyroids, and that there is a strong tendency for this to become malignant.

In 45 cases[12, 13, 14] collected from the literature, and including our own, the condition became obvious in twenty-five before the age of 30, and in fifteen before the age of 20 years.

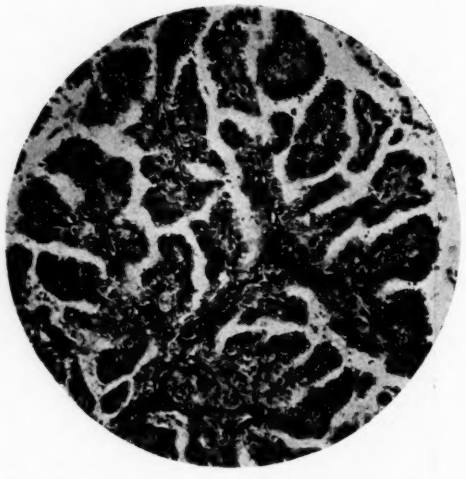

FIG. 89.—*Case* 4. Histological picture of one of the nodules shown in *Fig.* 88.

In the majority of these the change

was advanced when the pathological examination was made. The average age at which these tumours develop is far earlier than is usual with epithelial tumours of any other organ, but it does accord with the age at which non-malignant enlargements of the thyroid gland occur—the enlargements which we regard as compensatory or resulting from some kind of stimulation.

Papilliferous change is almost universal in lateral aberrant thyroids. It was present in our four cases, and in practically all the published cases at the time the patient was first seen. (Mr. Cecil Joll has very kindly shown me a section from a tumour removed by the late Mr. Charles Ryall in which papilliferous change had not occurred.)

The early age (adolescence in some) of the patients, and the very high frequency of the papilliferous change, almost compel the question whether the gland tissue of lateral aberrant thyroids does not undergo papilliferous hyperplasia under the stimulation of the ordinary physiological demands.

In the body there is always interaction—and usually balance—between stimulation and capacity for response, but in the thyroid gland we frequently see this equilibrium disturbed, on the one side through variation in the intensity of the stimulation, and on the other through variation in the capacity of the organ to respond. If the physiological demands of the body could be kept at a lower level, the lateral aberrant thyroids might remain as imperfectly migrated 'rests'. If the gland tissue of which they consist were more completely developed, it might still function without tumour formation. In this respect it is interesting to revert to the tumours occurring in brook trout, where a condition in many ways so nearly approaching carcinoma can be induced in this very labile organ by the appropriate stimulation.

MALIGNANT ADENOMA.

'Malignant adenoma' is not a perfect term, but in the present state of knowledge it would appear to be the most satisfactory. It is meant to convey that malignant transformation has occurred in an adenoma, and that in some respects the tumour maintains the morphological characters of an adenoma; and by the term 'adenoma' is meant the benign neoplasm usually called in this country 'fœtal adenoma'. Aschoff[5] has shown that adenomata arise from proliferation of adult cells, not from fœtal rests as was formerly believed. The capsule is formed by condensation of the stroma, and by compression of neighbouring tissue. The term 'fœtal' is therefore not correct if it is meant to indicate the origin of the tumour. It may be used to indicate that the tissue is more embryonic in appearance than normal gland.

This type of carcinoma constitutes a high percentage of the total numbers. If an average is taken from large clinics it includes 85 to 90 per cent of the total.

Benign adenomata differ from one another in histological appearance, though they are always clearly distinguished from involution nodules. Some are composed of anastomosing columns of cells (*Fig.* 90). In others differentiation has occurred, and the tumour consists of follicles. Many of these are immature, containing no colloid, and scarcely a lumen, while other follicles are more mature and contain colloid (*Fig.* 91).

When malignant transformation has occurred, these ancestral characteristics are maintained to a considerable extent, although ultimately—early in some, but very late in others—differentiation is lost and the cells grow

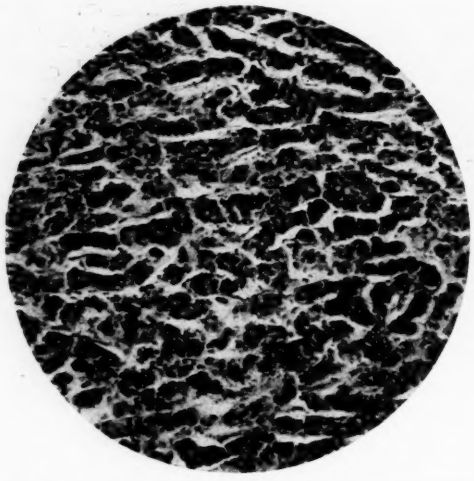

FIG. 90.—Benign adenoma showing anastomosing columns of cells forming miniature tubules.

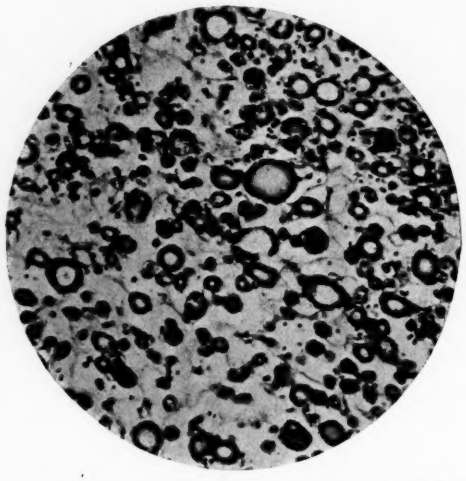

FIG. 91.—Benign adenoma. Some vesicles contain colloid, some have no lumen, others are quite immature.

in disorderly fashion. The histological appearance of malignant transformation that has taken place in the first type of adenoma is shown in *Fig.* 92. This is from a deposit under the skin, but superficial to the sternomastoid muscle, in the scar of the operation for the removal of the adenoma. In this the anastomosing columns are still clearly seen, although growth is more active. It may be asked: Is this really malignant? *Fig.* 99 shows deposits in the lungs in this patient.

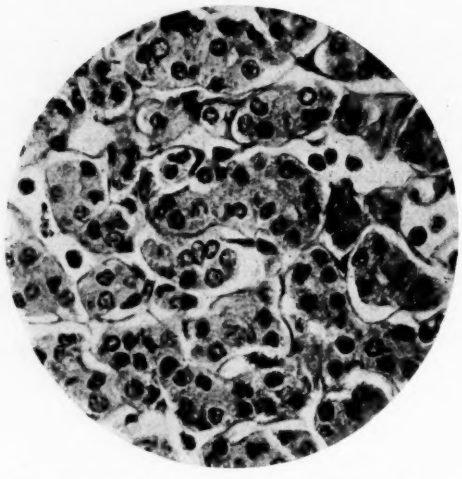

FIG. 92.—Malignant adenoma arising from a tumour of the type shown in *Fig.* 90.

An example of the second type—that is, the retention of the follicular structure of the parent adenoma after malignant transformation had taken place—is illustrated by *Fig.* 94. Again it may be asked: What are the criteria of malignancy, for the follicles in some parts are as highly differentiated as in some non-malignant adenomas? In this patient the growth has penetrated veins and filled the large vessels on the surface of the gland; not only so, but it has transgressed the capsule and invaded the tissues of the neck. A blood-vessel completely filled with tumour growth is shown in *Fig.* 93. The growth filling this vessel retains its follicular structure (*Fig.* 95).

These morphological differences, together with some biological characteristics, have led Continental observers to magnify subdivisions of malignant adenoma into definite types of carcinoma under the names of 'proliferating adenoma' and 'metastasizing adenoma'. This nomenclature appears to be unnecessarily complicated and mystifying, for in malignant adenoma every cellular type is found between a high degree of differentiation on the one hand and complete de-differentiation or anaplasia on the other. To a great extent the degree of differentiation is in inverse ratio to the grade of malignancy. When differentiation is lost, the highly cellular growth gives the appearance conventionally called 'medullary' (*Fig.* 96), and many authors have given this name to a type of carcinoma. This also is unnecessary. It does indicate a grade of anaplasia, but not a different class of carcinoma.

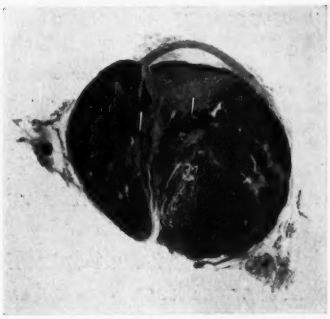

FIG. 93.—Section of a vessel from the surface of the tumour illustrated in *Fig.* 94. (× 3.)

Malignant adenoma not only differs morphologically from papilliferous adenocarcinoma, but its method of dissemination is different. We have seen that papilliferous adenocarcinoma disseminates primarily to adjacent lymph-nodes. Malignant adenoma does not disseminate to lymph-nodes while

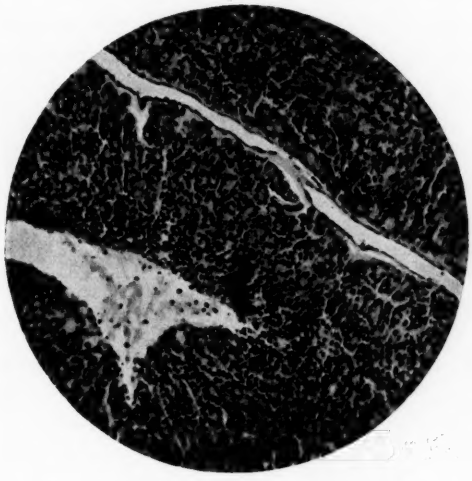

FIG. 94.—Malignant adenoma retaining follicular structure.

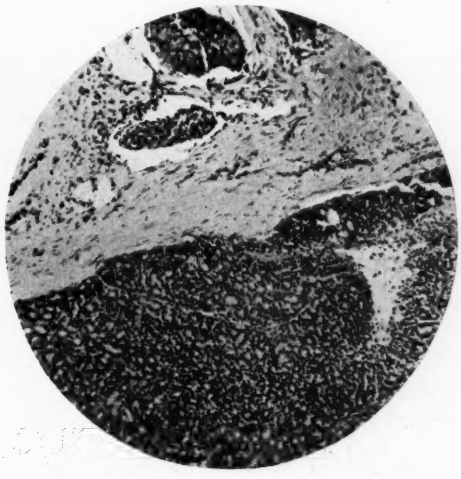

FIG. 95.—Microphotograph of growth filling the vessel shown in *Fig.* 93.

contained within its capsule, but even at this stage it invades blood-vessels, and therefore distant metastases may occur while the primary growth is small. This has given rise to the theory, so long believed, that metastases could originate from a normal thyroid gland, or from small benign adenomata. Invasion of blood-vessels is almost certainly responsible for the deposits seen in *Fig.* 99. As the percentage of cases of carcinoma of the thyroid gland originating in benign adenomata is so high, it is important to try to

decide when the change is taking place. *Fig.* 97 is a microphotograph of a section of an adenoma removed from a patient in whom malignancy was unsuspected, the nodule having been present eight years and enlarging

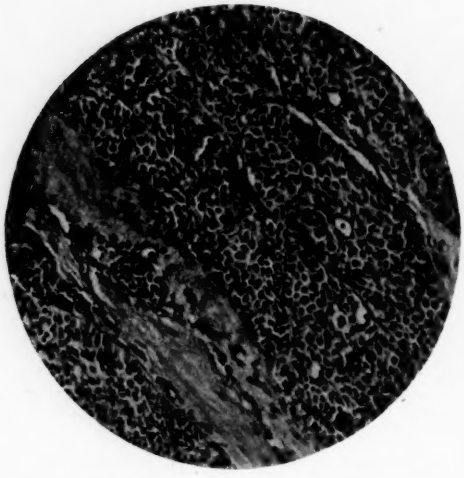

FIG. 96.—Malignant adenoma in which structure has been lost.

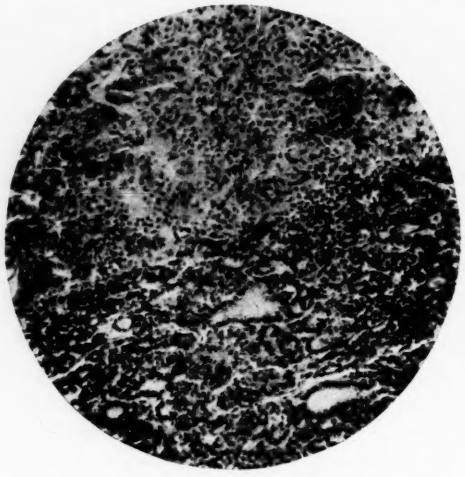

FIG. 97.—Microphotograph of section of an adenoma removed eighteen years ago. It was then considered to be suspicious of malignancy.

slowly. The section is not very clear, for it was cut and stained eighteen years ago and with no idea of reproduction. The cells appeared to be unduly active for an innocent tumour, but could not be diagnosed as definitely malignant. The tumour was classed as suspicious, and the patient kept on a follow-up list. Five years later a nodule appeared under the skin in the scar over

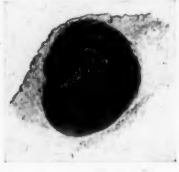

FIG. 98.—Nodule removed from the extreme end of the incision, over the sternomastoid muscle. The skin is seen superficial to it. This is from the same patient as *Figs.* 92, 97, 99.

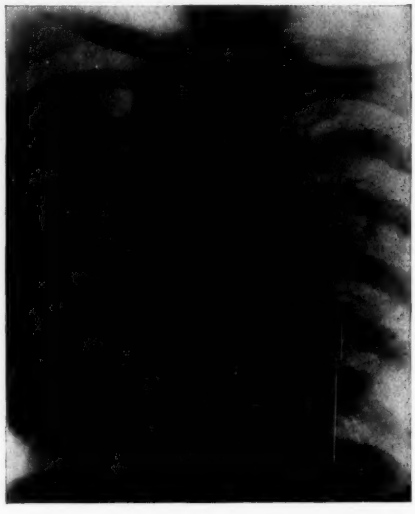

FIG. 99.—Deposits in both lungs. From the same patient as *Figs.* 92, 97, 98.

the sternomastoid muscle. Ten years from the time of removal of the original tumour a similar nodule appeared. *Fig.* 98 shows the whole nodule with the skin stretched over it, and *Fig.* 92 the histological appearance. Four years later—fourteen years from the original operation—severe hæmoptysis led to an X-ray examination of the lungs being made. Deposits were found to be present in both

lungs (*Fig.* 99). Therefore unusual activity of epithelial cells should put us on guard and make us watch for biological evidence.

The varied changes that may occur in one adenomatous nodule are well illustrated in *Figs.* 100–103. They are from a patient in whom a tumour had

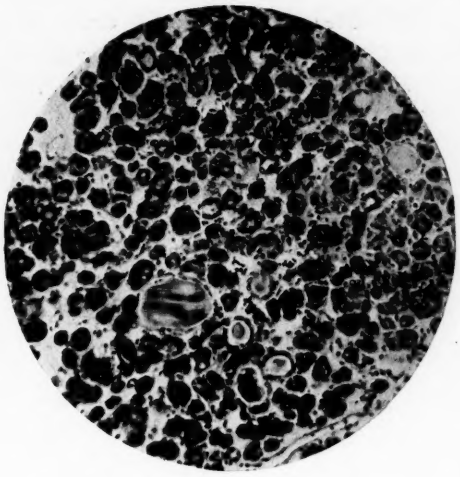

FIG. 100.—This section by itself is consistent with the tumour being a benign adenoma.

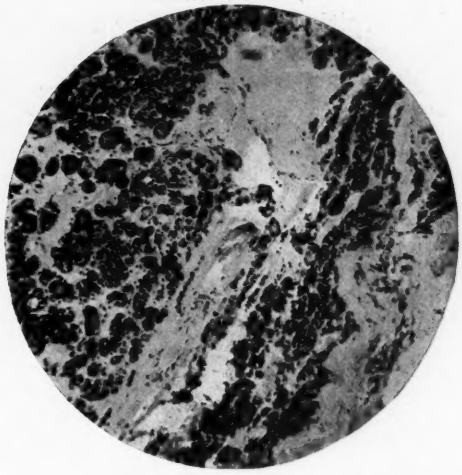

FIG. 101.—The epithelium, while retaining its follicular arrangement, is invading and penetrating through the capsule. From the same case as *Figs.* 100, 102, 103.

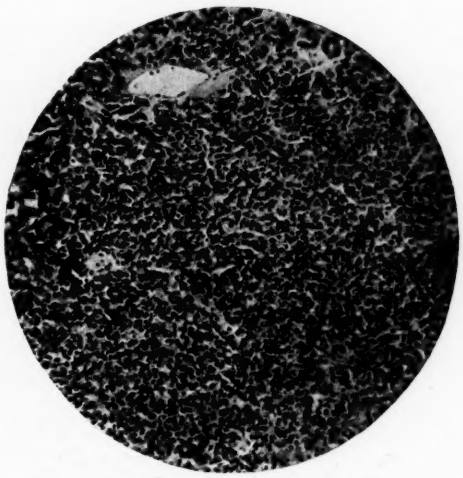

FIG. 102.—This is still suggestive of vesicle formation, but structure is practically lost and the epithelium more embryonic. From the same case as *Figs.* 100, 101, 103.

FIG. 103.—A small area of papilliferous growth occurring in the same tumour as *Figs.* 100–102.

been present for eighteen years and shortness of breath for one year. The enlargement had been present so long that she had become accustomed to it. The first (*Fig.* 100) shows a typical 'fœtal adenoma'. Many of the vesicles are well formed and contain colloid. Others are immature and without lumen. *Fig.* 101, from the same patient, shows invasion of the capsule even while

the cells are retaining their follicular arrangement. It may be objected that the capsule is growing in and strangling the tumour. I think that this is not so, for the nuclei all stain well; also the following sections from other parts of the tumour give confirmatory evidence.

Fig. **102**, from another part of the same tumour, shows the arrangement of the epithelium becoming much more active and embryonic in character.

FIG. 104.—Adenomatous nodule.

FIG. 105.—Cross-section of the nodule shown in *Fig.* 104.

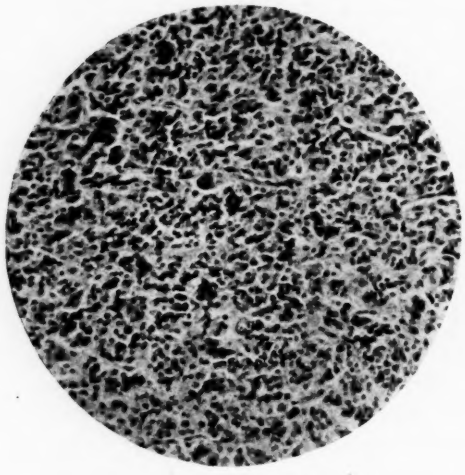

FIG. 106.—Microphotograph of one area of the nodule in *Fig.* 104, showing malignant change.

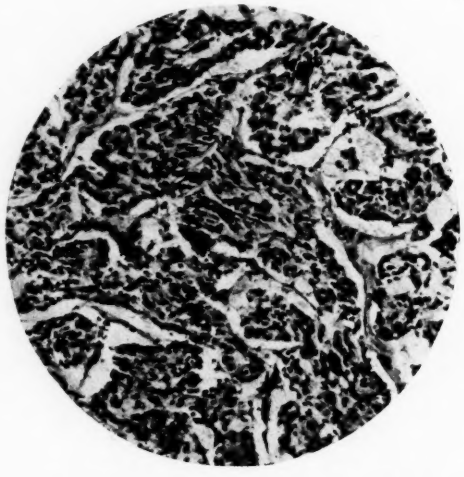

FIG. 107.—Microphotograph of another area of the nodule in *Fig.* 104, showing malignant change with different characters.

Such vesicles as exist are immature, contain no colloid, and many contain no lumen. The epithelium is breaking away from vesicle formation altogether. In still another part papilliferous growth has occurred (*Fig.* **103**). Add to all this epithelial activity the fact that this tumour—not large in itself—could not be dissected off, but had to be cut away from the upper rings of the trachea and the thyroid cartilage, and I think there can be no doubt that malignant change had taken place.

The change within the capsule may be very extensive before clinical evidence of invasion becomes obvious, or any suspicion of malignancy is aroused. The nodule shown in *Fig.* 104 had been known to be present in the thyroid gland of the wife of a medical man for six years. Its lower margin just dipped beneath the left sterno-clavicular articulation. It could be freely moved, but it had begun to cause pressure symptoms, and it was conspicuous. The patient disliked the look of it, and her husband realized that the pressure symptoms were interfering with her comfort. It was cleanly removed. On cutting across the specimen, suspicion was aroused (*Fig.* 105), and on microscopical examination very active malignant change was revealed (*Figs.* 106, 107).

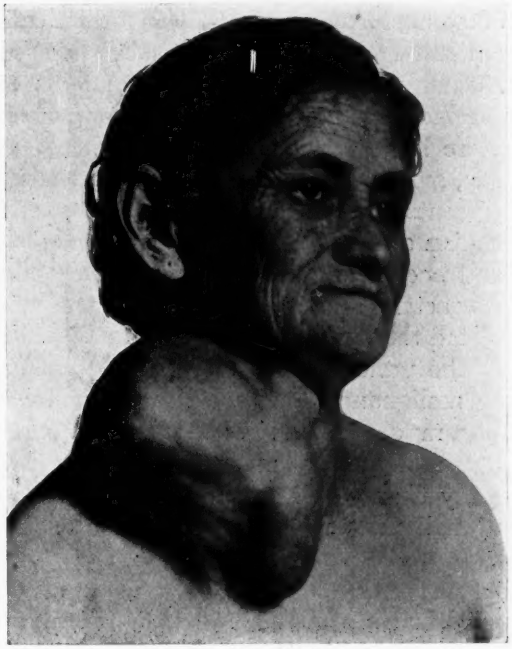

FIG. 108.—Carcinoma of thyroid, malignant adenoma type.

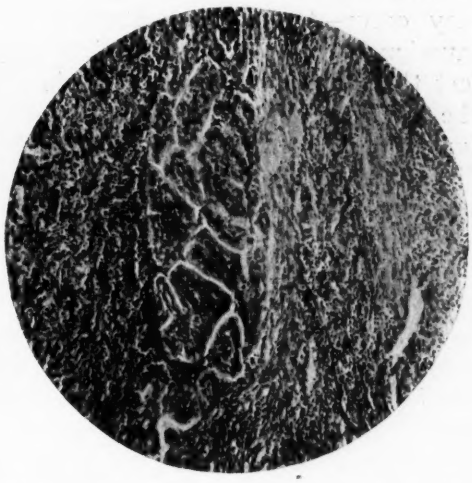

FIG. 109.—A small papilliferous area in the tumour shown in *Fig.* 108.

Advanced Cases.—I have not considered advanced cases of malignant adenoma. That would serve no useful purpose at this part of the discussion. In the late stages of the disease specific glandular structure will generally have disappeared, and the histopathological appearance will be that shown in *Figs.* 96, 111, and 113. When discussing classification it was stated that the second and third types of carcinoma tend to overlap. This is shown in *Fig.* 103, where in a characteristic malignant adenoma one papilliferous area was found. Thyroid epithelium seems never to lose this capacity entirely. In the case shown in *Figs.* 108, 109 the growth consisted of sheets of cells which in many places were suggestive of sarcoma; yet in one microscopic field a tiny area of papilliferous formation is seen. This rarely prevents each type from being readily classed. Again, in the later stages of papilliferous adenocarcinoma an area will sometimes be found where the epithelium is growing in broad sheets (*see Fig.* 77). The classification must not be made too rigid.

Discussion on the Origin and Pathology of Malignant Adenoma. — The origin and evolution of adenomata has been described by Aschoff.[5] From his detailed description it cannot be doubted that they occur as the result of some form of stimulation or irritation. In a small percentage of these, epithelial activity crosses the line dividing innocency from malignancy. Although this percentage is small, it constitutes by far the biggest class of carcinomas of the thyroid. How long the epithelial activity has been taking place before any evidence of it becomes obvious clinically it is impossible to say, and it will have been realized that the interpretation of the histopathology presents great difficulty. This is because the epithelial appearance of different benign adenomas, or different parts of the same adenoma, may vary so widely, and because malignant change may take place with so little alteration in the characters and arrangement of the epithelium. Graham and Wegelin have stated that neither the character of the cells, nor the mitoses, nor the structure of the vesicles admits of any definite conclusion as regards non-malignancy or malignancy, and we heartily agree with the latter when he states that "from a pathological standpoint the dividing line between non-malignant and malignant tumours can probably nowhere be drawn with greater difficulty than just here."[20] Graham[21] considers vascular invasion the chief test of malignant change in an adenoma. Sometimes (*see Figs.* 93, 95) the invaded vessels are obvious on the surface of the growth; sometimes they are only found after patient search under the microscope. In malignant adenoma neighbouring glands are not infected while the growth is contained within the capsule—in this respect it differs from scirrhus and papilliferous adenocarcinoma—but the invasion of veins may occur very early and while the growth is still encapsulated. As a consequence of this, distant metastases may occur—to lungs or bones—while the parent growth is small. This invasion of veins while the primary growth remains small, together with the fact that secondary deposits retain the characters of the primary tumour, is the explanation of the statement that metastases may arise from benign tumours. We have not had an example of an *inconspicuous* tumour giving distant metastases, but we have of a tumour giving rise to lung metastases while no invasion of lymph-nodes in the neck was ever found (*see Fig.* 99).

In spite of the difficulty sometimes found in interpreting the histopathology, I believe that the sections presented have shown changes that it is impossible to ignore. The search must be thorough, and sections taken from several places. Remembering the various types of epithelial arrangement that may occur in non-malignant tumours, all the evidence must be considered, including epithelial activity, mitosis, the beginning of alteration in structure, sometimes invasion of capsule or vessels even when specific glandular structure is retained. Seeing that the question is not a purely academic one, since we are dealing with human beings, all clinical evidence must be taken into account.

CLINICAL COURSE OF CARCINOMA OF THE THYROID GLAND.

1. **Scirrhus or Carcinoma Simplex.** — This type does not differ in its habits from the same type of carcinoma occurring elsewhere.

2. Papilliferous Adenocarcinoma.—The evolution of this type is without doubt slow in the majority of cases, but it is inevitably progressive. Sir James Berry[22] wrote that these tumours grow slowly and are more amenable to treatment by removal, even though they may have attained large size. He cites Mr. Barker's patient[23] who lived eighteen years after the first appearance of the tumour. This patient underwent numerous operations, but the growth caused his death in the end. The fact that growth is slow does not make these tumours any the less malignant. Among the cases illustrated it will be remembered that even in a papilloma regarded as benign the epithelial activity was suspicious, and that in successive cases invasion of adjacent tissues and deposits in lymph-nodes occurred.

FIG. 110.—Malignant adenoma, intrathoracic. Skiagram taken ten years after the removal of the original tumour from the neck. This tumour was removed after splitting the sternum. Recurrence in the chest took place quickly.

3. Malignant Adenoma.—In this condition it has generally been assumed that growth is more rapid, but even in this type it varies within very wide limits. One patient died within twelve weeks from the time she first recognized that her adenoma had begun to cause discomfort, while another is alive eighteen years after removal of a tumour diagnosed as malignant, and proved by the subsequent history of the patient to have been so (*see Figs.* 92, 98, 99). The following case lies between these two extremes:

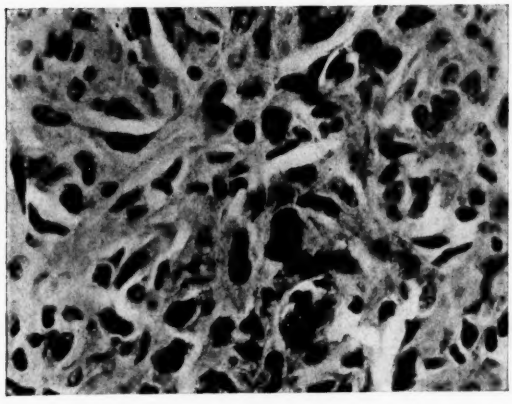

FIG. 111.—Microphotograph showing histopathology of tumour shown in *Fig.* 110.

A mass had been removed from the neck on account of extreme dyspnœa ten years previously in another hospital, and it was pronounced malignant by the hospital pathologist; that the diagnosis was correct was proved by the later history. The mass removed by me at this late stage was undoubtedly malignant (*Figs.* 110, 111). Recurrence took place, and the patient subsequently died, but ten years had elapsed before the tumour began to grow rapidly.

In the patient from whom the sections shown in *Figs.* 93–95 were taken the tumour had been present for fifteen years. Some increase in size one year previously had not disturbed him, but he began to be rather irritated by the constant necessity for clearing his throat of phlegm and the difficulty in doing so. Large surface vessels were invaded with growth. The tumour

from which *Figs.* 100–103 were taken had been present for eighteen years. It is not possible to know when the change had begun in the last two cases, but in the two preceding these there is microscopical evidence of epithelial activity extending over eighteen and ten years respectively. I therefore feel sure that in many cases malignant adenoma is comparatively slow in its evolution. In its earliest stages it is confined within the capsule of the adenoma, and how long it is from the time the first change begins in the epithelium until the capsule is penetrated, it is impossible to say. Certainly it will have begun before increase in size of the nodule is obvious. It precedes—for it causes—the pressure symptoms and fixation. The first suspicion of change will be aroused by clinical signs, and then all the evidence should be considered. If the tumour is removed, the most careful examination must be made, and for this blocks of tissue must be chosen from several parts of the tumour. Often enough the diagnosis is dated from a period subsequent to penetration of the capsule, when the induration, the nodular appearance, and the pressure symptoms leave no doubt of the condition.

PROGNOSIS.

Malignant disease of any kind offers no cheerful outlook, and malignant disease of the thyroid gland has been regarded rather hopelessly. Looking through some recent text-books I find that operation is only advised in the early stages—that is, when the outline is still clear, mobility present, and enlarged glands or secondary deposits are absent; also the mortality of operation is placed as high as 25 to 30 per cent; that most cases are inoperable when first seen, and that radium and X-ray treatment have little effect on the growth; that the growth should still be within the capsule of the gland and must not be adherent to the trachea; and that (when commencing in an adenoma), if local malignancy is clearly shown by signs, no operation should be done, but if there is doubt, operation should be performed on the chance of a cure.

It is true that in scirrhus any lasting benefit is unlikely to be obtained; but scirrhus is rare, and even in this type it is a hopeful sign that the tumour from which *Fig.* 66 was taken was temporarily reduced in size by at least half by the use of radium needles. In the two other types—and they constitute the vast majority—the outlook should be less gloomy for the following reasons:—

1. Diagnosis is made—or suspicion aroused—much earlier than was the case formerly.

2. The rate of growth in papilliferous adenocarcinoma is slow. In many cases of malignant adenoma it is also slow, and is for a long time retained within the capsule. (It is to be borne in mind that dissemination by the blood-stream may occur before invasion of the capsule.)

3. Experience is showing that malignant epithelial cells of thyroid origin are radio-sensitive. I say this after making every allowance for error of judgement on account of the slow rate of growth in many cases.

To inform a patient with a hard nodule that it is unimportant and should be ignored is not right. It is not our function to operate upon every

abnormal thyroid, but when our advice is sought regarding a lump, it is our function to weigh all the evidence and to advise the patient wisely.

Again, even in late cases, it is possible to plan and carry out an operation which may give great relief in itself and may make further relief possible by X-ray treatment.

In our series no patient has died at operation, although in some the condition has been advanced, causing the patients great distress. In several patients the sternum has been split to obtain access to a deep thoracic mass (*Figs.* 110, 112), and in others a large fixed mass has surrounded the neck, involving the deep structures and invading the upper inlet of the thorax to an extent that made breathing extremely difficult.

FIG. 112.—Malignant adenoma invading the superior mediastinum. The greater part of this was removed after splitting the sternum.

RADIO-SENSITIVITY.

X RAYS.

The first patient in whom the value of X-ray treatment was brought home to me was seen in 1922. The patient had a large tumour encircling the front of the neck, with extension into the thorax. The extent of the intrathoracic growth is shown in *Fig.* 112, and with the massive growth in the neck it will be realized that respiration was greatly hampered. Because of the fixation of the growth, access to the intrathoracic portion could only

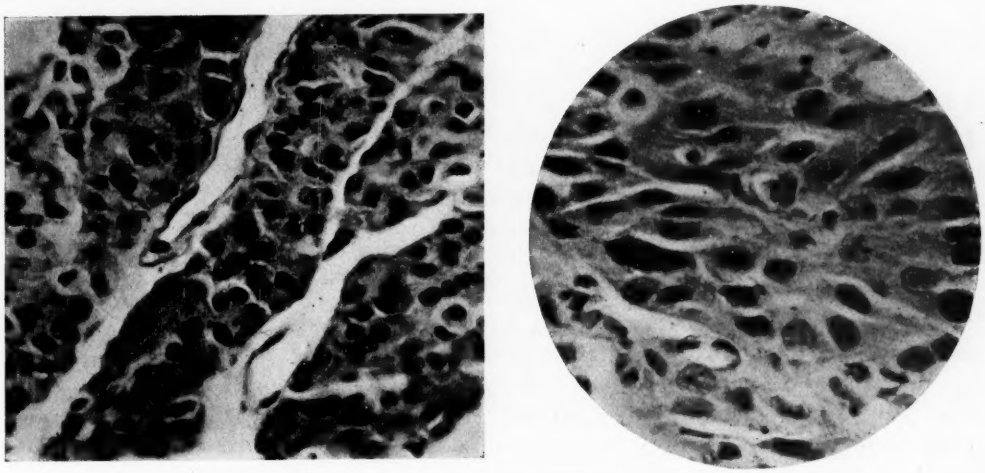

FIG. 113.—Microphotographs from two portions of the tumour removed from the patient whose skiagram is shown in *Fig.* 112.

be obtained by splitting the sternum. Growth was cut across both in the thorax and in the deeper planes of the neck. The appearances of the tumour are shown in *Fig.* 113. It was of malignant adenoma type. Because of the growth known to be left, X-ray treatment was given, though this was not deep X-ray therapy in the modern meaning of the term. A subsequent skiagram (*Fig.* 114) shows the contrast in the upper thorax. The patient resumed his work and carried it on for two and a half years. Then secondary deposits in the skull revealed themselves, and cachexia and death followed. The growth was a malignant adenoma of medullary type.

In another patient, seen in 1926, the growth was a malignant adenoma. It had transgressed the capsule, and was firmly fixed. The histopathology is shown in *Fig.* 96. Dyspnœa and dysphagia were severe. She could only

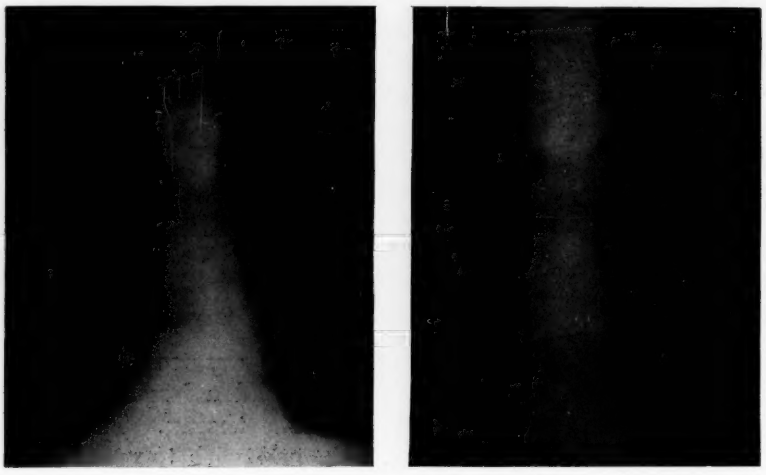

FIG. 114.—The appearances of the superior mediastinum before and after excision of the tumour shown in *Fig.* 112, and subsequent X-ray treatment.

swallow soft foods and liquids. Local recurrence followed operation. Deep X-ray therapy appeared to bring about disappearance of the recurrence. This patient died four years later from cerebral abscess. A small area of growth had eroded the common carotid artery.

Another patient, operated upon in 1927 for malignant adenoma (*Fig.* 115), is still alive and apparently well, except for paralysis of one vocal cord. This was due to damage to the nerve at operation, for the growth was extensive and much dissection was necessary.

These cases were of malignant adenoma type. The two following are examples of the result obtained in the papilliferous adenocarcinoma type.

Figs. 79–81 are from a patient with carcinoma in the thyroid gland and deposits in the lymph-nodes of the neck, seen and operated upon in 1924. The late Mr. Barker and Sir James Berry in this country have kept patients alive for many years by multiple operations, but the disease ultimately resulted in death. More recently cases have been reported in which, when the disease has been operated upon early the patients are still alive.[3]

In the case under discussion, diagnosis was not made early. The patient underwent two operations, but lymph-nodes continued to enlarge. X-ray treatment was commenced at St. Bartholomew's Hospital, and the nodes disappeared. Now, seven years subsequently, this patient is robust, there is no sign of growth, and for several years she has been at full work.

Fig. 116 is a microphotograph from a patient who was seen and operated upon in 1924. An irregularly shaped tumour of the thyroid was present. It had penetrated the capsule and become fixed to the overlying structures. Dyspnœa compelled her to seek treatment. The greater part of the tumour was removed, but its removal was known to be incomplete. The illustration shows groups of carcinoma cells lying between muscle bundles. X-ray treatment was given. Now—seven years later—she appears to be perfectly well.

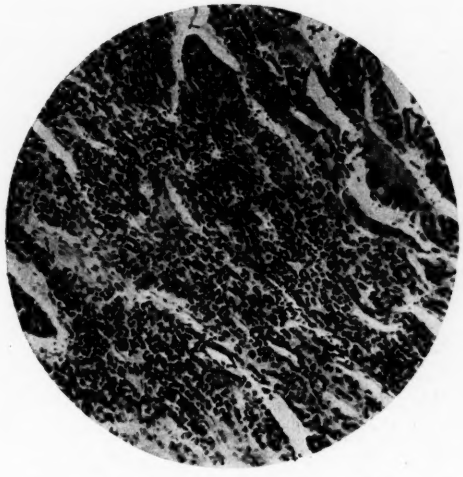

FIG. 115.—Malignant adenoma. Excision of tumour and subsequent X-ray treatment.

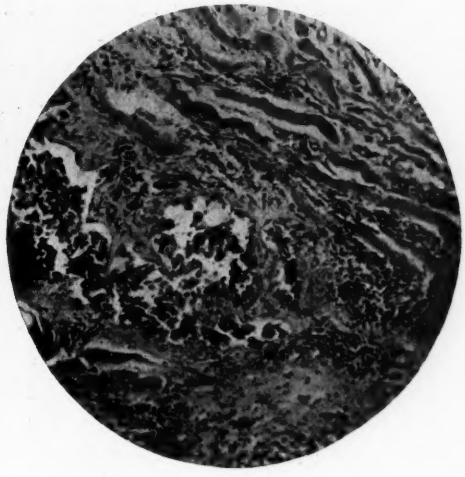

FIG. 116.—Carcinoma cells invading the overlying muscles. This patient is well seven years after treatment was commenced.

In 14 cases X-ray treatment has been of decided benefit. Of these, 10 are still alive: 2 seven years after commencement of treatment, 2 five and a half years, 3 three years, 1 two years, and 2 others almost one year after commencement of treatment. These latter are too recent to be used as evidence.

In judging the result of X-ray treatment, the slow rate of growth in many cases must be remembered, but it can scarcely be doubted that in some of them a stage had been reached that was becoming incompatible with life. With reference to the slow rate of growth, something should be said of the patient mentioned earlier with the long history culminating in deposits in both lungs (*see Figs.* 92, 97–99) and severe hæmoptysis. No X-ray treatment had been given in the earlier years of this patient's history. The original tumour was apparently completely removed within its capsule, but —the subsequent history shows—not before cells had reached the lungs through blood-channels. Fourteen years later these had developed into multiple tumours causing severe and repeated hæmoptysis. Deep X-ray

therapy was begun at once. The hæmoptysis ceased, the patient has returned to Australia, and there have been no attacks of hæmoptysis since —that is, for three and a half years. It is possible, but scarcely likely, that when these deposits in the lungs had reached the stage of causing hæmorrhages, the hæmorrhages would have ceased spontaneously. There are ten of these patients not only alive but apparently perfectly well. Instead of needing multiple operations, the signs of the disease have disappeared, and no recurrence has taken place.

These results are not always obtained. It might be expected that penetration by X rays would be such that cases would respond whether operation had been performed or not. Our experience has been that where a large tumour has been present and none of it removed by operation, no improvement has followed X-ray treatment. In three cases no improvement has followed even after removal of the bulk of the tumour.

RADIUM.

Only two cases have seemed to be suitable for radium treatment. In one needles were implanted after the removal of a tumour which microscopical examination proved to be a papilliferous adenocarcinoma, and which was imperfectly removed. In the other (*see Fig.* 66) removal proved impossible because of dense fixation of a scirrhous carcinoma. No improvement followed X-ray treatment. Radium needles were then implanted (*Fig.* 117). Following this the tumour diminished in size by at least half. It subsequently increased and the patient died. The reduction in size was striking, and it shows that the epithelium in scirrhous carcinoma is radio-sensitive.

FIG. 117.—Radium needles implanted in carcinoma of the thyroid (scirrhus). This is the patient from whom *Fig.* 66 was taken.

The use of radium by the insertion of needles presents difficulties. It is impossible to tell how deeply the growth has extended in the neck or into the thoracic inlet. It is likely to involve structures which it would be dangerous to puncture with needles.

In our experience the best treatment at the present time is to remove all that can be safely removed, and then have X-ray treatment given. The results even in advanced cases have often been surprisingly good.

DISCUSSION.

Leaving scirrhus out of this survey, for nothing has been observed which adds to our knowledge of it, we have evidence of epithelial hyperplasia

occurring in papilliferous adenocarcinoma, in lateral aberrant thyroids, and in malignant adenoma. The first two of these have so many points in common that they would seem to be practically similar conditions. Is malignant adenoma essentially different? We know that the hyperplasia which thyroid epithelium undergoes in its attempt to cope with demands made upon it, or as a response to stimulation, may be either papilliferous or follicular in type. Then we remember Aschoff's work where he points out that benign adenomata originate as tiny areas of epithelial proliferation evidently as a result of irritation or stimulation. The proliferation continues with gradual increase in size until the encapsulated nodules which we recognize clinically are produced. The majority of these remain benign, and many degenerate. In the few in which epithelial activity continues and invasion of vessels of the capsule occurs, we should like to believe that this is the result of continuance of irritation or stimulation. It does not seem unreasonable to suppose that the same factors are at work and analogous effects brought about in malignant adenoma and in papilliferous adenocarcinoma.

The investigation of our material has compelled me to place our cases in a sort of linear series by which we pass from degrees of hyperplasia which may resolve by appropriate treatment, to benign neoplasms, and finally to invading and disseminating growths. The gradation between each member of the series which along one line leads to papilliferous adenocarcinoma, and along the other to malignant adenoma, is so insensible that I am persuaded that we are confronted with a series of responses not different in kind, but only differing in degree. That there is some connection between functional capacity and this hyperplastic response is suggested by the frequency of the papilliferous change and the period at which it takes place in lateral aberrant thyroids, the tissue of which is probably imperfectly developed and therefore of low functional value, and by the higher incidence of malignant adenoma in centres of endemic goitre where the affected glands are subefficient.

Although carcinoma of the thyroid is rare in comparison with its occurrence in some other organs such as the breast and the uterus, and therefore material for research is scarce, and although in the early stages the difficulties of drawing the line dividing innocency from malignancy are so great, this organ presents some unusual advantages in the attempt to unravel the difficulties. Among these advantages is the extreme responsiveness of the thyroid gland to the demands of the organism in human beings, and the wide range of these demands (varying from puberty to grave psychic disturbance, and possibly endocrine imbalance), while in animals—as in brook trout—the gland may be subjected to experimental observation. This responsiveness is such that deviations from the normal are quickly recognized and are reflected in the histology of the gland. Apart from malignancy, many of its diseases are, for the moment, dealt with surgically, and therefore much material is available for the investigation of the histology of its very varied pathological conditions, as well as of its chemical and biological activities. This gives opportunity to correlate the work on innocent and malignant tumours of the thyroid gland with that of Lenthal Cheatle[24] on "The Primary Tumour in Breast Carcinoma", of Sampson Handley[25] on

"The Menace of the Papilloma", and of Robert Muir[26] on "The Intra-epithelial Growth of Carcinoma" (preceding invasion of the basement membrane). It should also enable us clinically to avail ourselves of the work of Marine and his co-workers, of McCarrison, Mellanby, and others, who have made such valuable contributions to the animal experimentation side of this problem.

SUMMARY.

Pathological.—

1. In our cases there are three types of cancer of the thyroid: scirrhus, papilliferous adenocarcinoma, and malignant adenoma.

2. Scirrhus does not differ from the same disease occurring elsewhere in the body.

3. In the thyroid gland epithelial proliferation is the characteristic response to stimulation. The proliferation may resolve spontaneously or under treatment; it may form a benign tumour, or it may form a tumour which invades and disseminates. These stages merge into one another by insensible gradations. These histological gradations cause difficulty in deciding just when a tumour has become malignant.

4. Proliferation of thyroid epithelium may be papilliferous or follicular in type. Papilliferous adenocarcinoma may be an ultimate result of the former, malignant adenoma of the latter type.

5. It is suggested that the cause of carcinoma is stimulation, which may be normal and affecting tissue which is sub-efficient, or so excessive in degree as to be abnormal and amount to irritation.

6. Although in cases of carcinoma of the thyroid specific glandular structure is frequently retained both in the parent growth and in the metastases, the essential character of the disease conforms to the same laws observed in carcinoma of other glandular organs, and its cause is possibly the reaction of the glandular epithelium to irritation as suggested by other workers in, for example, carcinoma of the breast.

Clinical.—

1. A nodule in a thyroid gland should not be regarded as of no importance, and early changes in the signs or symptoms associated with it should induce the practitioner to investigate the cause of these changes.

2. Histological examination should be made in every case of removal of thyroid tissue, and sections should be taken from different areas.

3. Advanced cases should not be regarded as hopeless. When the condition of the patient justifies it, as much of the tumour should be removed as possible, and then X-ray treatment commenced. By this means comfort is given, and life is sometimes prolonged to a surprising extent.

The sections shown in this article have been cut and the micro-photographs made by Miss M. Vaughan of the Surgical Professorial Unit, St. Bartholomew's Hospital. Some of the illustrations are appearing in the new edition of Choyce's *System of Surgery*, and these are reproduced by mutual agreement.

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THE PATHOLOGY AND TREATMENT OF TUMOURS OF THE CAROTID BODY.

By H. HAMILTON STEWART,

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The carotid body was first described by Neubauer in 1786, although Haller[1] had mentioned its presence many years previously. Since then many different names have been given to this apparently insignificant structure, according to whether it has been thought to be an endocrine gland or a ganglion. Following the work of Kohn[2] it was generally accepted that the carotid body was derived from embryonic ganglion cells of the sympathetic plexus, and was therefore included among the chromophil tissues. However,

Fig. 118.—Section of normal carotid body, to show various types of cells which are met with in this structure. Attention is specially drawn to the large cells with a darkly stained nucleus situated excentrically and finely granular and vacuolated cytoplasm. (× 600.)

de Castro[3] has recently produced evidence to show that the structure does not contain adrenalin; he suggests that the carotid body is related in some way to the carotid sinus.

I have dissected out a number of normal carotid bodies in the cadaver and examined them microscopically (*Fig.* 118). They are small pinkish bodies

situated in the middle of, or just deep to, the bifurcation of the common carotid artery on each side. The average size is $\frac{3}{16}$ in. long, $\frac{1}{8}$ in. broad, and $\frac{1}{16}$ in. thick. They have a definite capsule, and are attached to the bifurcation of the common carotid artery by a short fibrous pedicle. A branch of the glossopharyngeal nerve can be traced to this region. From the connective-tissue capsule fibrous septa enter and divide the structure into lobules, which are again subdivided by smaller trabeculæ of the same connective-tissue network. The organ is richly supplied with blood-vessels and nerves. The cells may be divided into two groups :—

Group A.—The majority are large polyhedral cells, rich in cytoplasm, which is finely granular and poorly staining. These cells occasionally form a syncytium. The nuclei are large, round or oval, somewhat excentrically placed, rich in chromatin, and show a distinct nuclear membrane. Other cells, resembling these, are found ; they are slightly larger and their cytoplasm, which is less granular, contains small vacuoles. The more vacuolated the cytoplasm, the less granular it becomes. These vacuolated cells are apparently derived from the granular cells.

Group B.—Many endothelial-like cells are to be found, particularly around the periphery of the lobules. They are small or medium-sized, have little cytoplasm and deeply-staining nuclei, which are oval or spindle-shaped. Occasional eosinophil tissue-cells are to be seen scattered about the structure, and fibroblasts are present in the connective-tissue framework.

CASES OF TUMOUR OF THE CAROTID BODY.

Case 1.—The patient, a carpenter, age 30, was admitted to St. Thomas's Hospital in February, 1928, under the care of Mr. Max Page.

History.—For four years the patient had suffered from attacks of sharp pain starting in the left side of the head, shooting down to the neck and to the left shoulder. In these attacks he fell to the ground, but did not think he lost consciousness ; in a few minutes he recovered and was able to get up and to continue his work.

On Examination.—There was a swelling about the size of a large walnut in line with the left carotid vessels, and situated at the bifurcation of the common vessel. It was fairly mobile laterally, but fixed vertically. The lump was firm in consistency and gave a transmitted pulsation from the main vessels. On firm pressure on the lump the man's pulse became slow and feeble. Dr. M. A. Cassidy obtained tracings which show the result graphically of the effect of pressure. No definite diagnosis was made.

Operation (Feb. 3).—A 3-in. incision was made along the anterior border of the left sternomastoid, and the tumour was exposed. The internal jugular vein appeared greatly distended, and it was necessary to divide it below the level of the tumour and strip it up before the relationships of the latter could be made out. The tumour was then found to be vascular and firmly adherent to the carotid vessels, so that resection of the latter was clearly necessary in order to effect its extirpation. Considering the severity and danger of the man's condition, it was decided that in this case extirpation was justifiable, even in the face of the great operative risk. After resection of a length of the jugular vein, the common carotid artery was divided half an inch below the tumour and, together with the latter, was stripped away from the underlying structures ; the vagus nerve was recognized and left intact. Part of the sympathetic chain was adherent to the posterior aspect of the tumour and had to be resected. Post-operative progress was uneventful, except for transient aphonia. A slight enophthalmos and definite miosis were noted

the day after operation and have persisted. The patient remains in perfect health at the present time and free from symptoms.

MACROSCOPIC APPEARANCE.—The tumour was ovoid and measured 4·5 by 3 cm. It was definitely encapsulated, and had a firm homogeneous structure on section; it was pale pink in colour. It was traversed by part of the common and the internal and external carotid arteries, to which it was closely adherent.

MICROSCOPIC APPEARANCE (*Fig.* 119).—The tumour was malignant (Professor L. S. Dudgeon).

Commentary.—I have found over a hundred cases of new growth of the carotid body recorded, but in only three of these was the tumour associated with syncopal attacks. In the light of recent physiological knowledge of the function of the carotid sinus, the case recorded above is of great interest. De Castro[3] and Heymans[4] believe that the carotid body is closely related to this

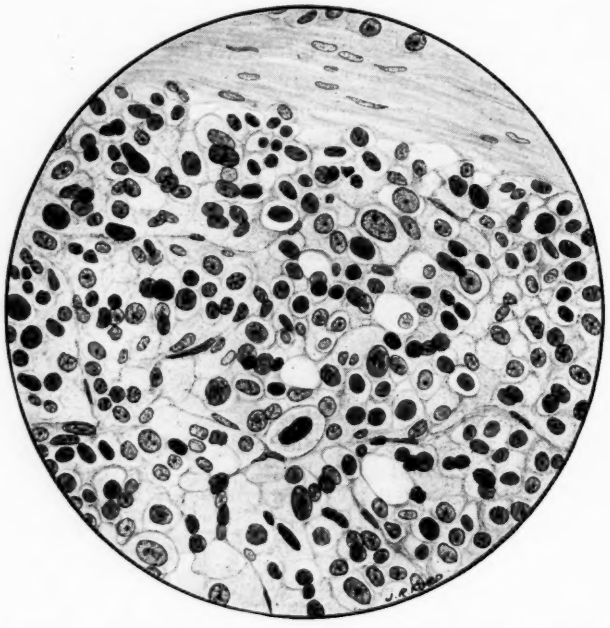

FIG. 119.—*Case* 1. Section of a malignant growth arising in the carotid body. This tumour, which is of the renal hypernephroma type, had invaded its capsule and extended into the lymphatic vessels. Note the appearance of some very large cells in the figure. (× 270.)

structure in function. In the physiological sense, the carotid sinus is a specially innervated part of the vessels in the neighbourhood of the bifurcation of the common carotid artery into its branches, i.e., the carotid bulb. This sinus and the carotid body are supplied by the glossopharyngeal nerve. By perfusion experiments it has been shown that stimulation of the carotid sinus, electrically or mechanically, provokes a combined reflex of cardiac inhibition and fall of blood-pressure, just as does stimulation of the central end of the depressor nerve. It is well recognized that pressure over this region of the neck causes slowing of the heart and syncopal attacks. Hering[5] has shown that this is not due to pressure on the vagus, as was previously supposed, but that it is due to pressure on this sinus. It is therefore easily

understood why pressure on the tumour produced the attacks in this patient. It is more difficult, however, to explain why the symptom is not produced in all cases.

Case 2.—A male patient, age 67, was admitted on June 10, 1929, to St. Thomas's Hospital, under the care of Sir Percy Sargent.

HISTORY.—The patient complained of a swelling on the right side of the neck of one year's duration. Hoarseness of the voice and some difficulty in swallowing had been noticed for a few months. He was otherwise healthy and did not complain of any pain.

ON EXAMINATION.—A firm, smooth tumour, of 2½ in. diameter, was seen on the right side of the neck at the level of the thyroid cartilage and in the line of the great vessels. It was not attached to the skin, but moved on swallowing and was apparently of thyroid origin. The trachea was displaced one inch to the left. The

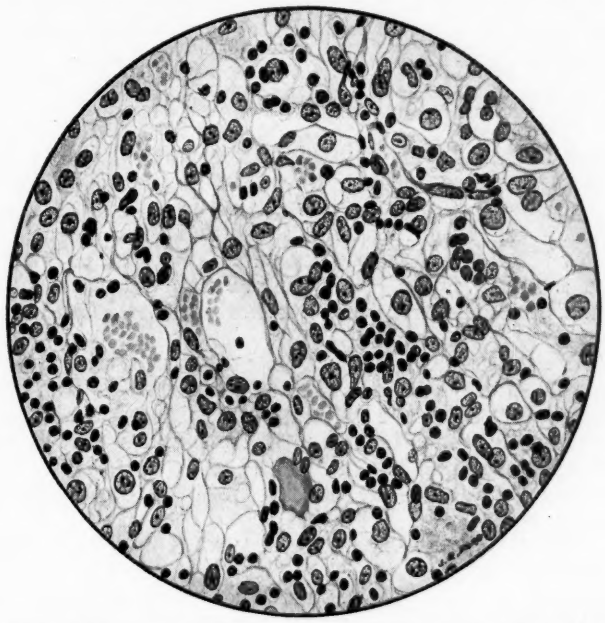

FIG. 120.—*Case* 2. Section of malignant growth arising in the carotid body which had invaded the sternomastoid muscle. (× 270.)

tumour was only slightly movable laterally, but was fixed vertically. Laryngoscopy revealed no abnormality.

OPERATION (June 19).—The tumour was found to be very adherent. The case was regarded as one of inoperable carcinoma of the thyroid gland, and a portion was removed for microscopy.

MICROSCOPIC APPEARANCE (*Fig.* 120).—Malignant tumour of the carotid body (Professor L. S. Dudgeon).

IRRADIATION.— On Aug. 26 the patient was re-admitted; the tumour had increased slightly since the previous admission. Thirty radium needles of 1 mgrm. each, screened by 0·5 mm. of platinum, were inserted into and around the growth through skin punctures and retained for a period of fourteen days. The tumour thus received an irradiation of 10,080 mgrm.-hours. A slight radium burn occurred. Following the irradiation the tumour decreased in size and swallowing became easier. The hoarseness of the voice persisted. On Oct. 27, 1930, fourteen months after the radium treatment, the tumour had reached its original size and was growing actively towards the mid-line. Hoarseness had increased and pain in the neck had been

present for three months. The tumour was still unattached to the skin, but was more fixed to the muscles. Slight movement still occurred on swallowing. The tumour was irradiated with 10 needles of 1 mgrm. each with the same screenage as before. A severe general and local reaction occurred, which necessitated the removal of the radium after eleven days' irradiation. On this occasion the radium treatment failed to have any beneficial effect. The patient was seen recently. He was very much weaker, in severe pain, and the tumour was growing rapidly. No evidence of any metastases could be found clinically.

Commentary.—The following points are of interest in this case: (1) The tumour on clinical examination appeared to be of thyroid origin. As the tumour was more actively growing towards the mid-line, it invaded the thyroid structures before becoming fixed to the other structures of the neck, and thus movement on swallowing occurred. As the growth progressed, the fixity became more general and movement on swallowing thus gradually diminished. (2) The effect of radium in this case was without permanent result. (3) Although the tumour was malignant, no evidences of glandular or general metastases were found clinically.

Case 3.—Mrs. G. W., age 41, a window-dresser, complained of a swelling on the left side of the neck, which had been present nine years. The tumour was painless and caused no symptoms. She was healthy.

On Examination.—A firm, smooth tumour was to be seen in the left side of the neck, just below the angle of the mandible. It measured $1\frac{1}{2}$ by 1 in., and lay in the line of the carotid vessels. The tumour was unattached to skin and was freely mobile from side to side, but fixed vertically. There were no other abnormal physical signs.

Operation (Feb. 27, 1929).—The tumour was exposed by Sir Percy Sargent through an oblique incision on the left side of the neck. It was completely encapsulated, and closely attached to the bifurcation of the left common carotid artery. The tumour was dissected free from the vessels and removed entirely without damage to nerves or main vessels.

Macroscopic Appearance.—The tumour was pinkish in colour, homogeneous, and completely encapsulated.

Microscopic Appearance.—A non-malignant tumour of the carotid body (Professor L. S. Dudgeon).

Subsequent History.—The patient made an uninterrupted recovery. She was examined recently and found to be entirely free from symptoms or recurrence.

Case 4.—A woman, age 70, had noticed a swelling on the left side of the neck for four years. It was painless and caused no symptoms, but recently had grown larger. The tumour measured 1 by $\frac{3}{4}$ in., situated at the bifurcation of the common carotid artery. It was thought to be an enlarged gland.

Operation (July, 1930).—The tumour was removed by Sir Percy Sargent without damage to nerves or main vessels. The patient made an uninterrupted recovery and there has been no recurrence.

Macroscopic Appearance.—The tumour was pink in colour, encapsulated, and had a homogeneous structure.

Microscopic Appearance.—The tumour showed no evidence of malignancy (Professor L. S. Dudgeon).

PATHOLOGY, DIAGNOSIS, AND TREATMENT.

Pathology.—

Macroscopic Appearances.—The tumours which were removed were pinkish in colour and encapsulated. On dividing them the structure was

homogeneous and closely resembled that of a fœtal adenoma of the thyroid. In *Case* 1 portions of the common, internal, and external carotids were embedded in the centre of the tumour. The malignant tumour in *Case* 2 was irremovable.

Microscopic Appearances.—I examined the microscopical sections of these four cases, and found that they were composed principally of cells resembling those of Group A (*see* p. 115) of the normal carotid body.

Non-malignant Tumours (*Cases* 3 *and* 4).—The large cells were arranged in more definite clusters than in the normal structure. These cell-groups were surrounded by young fibrous tissue. The cells had the same polyhedral form, and the cytoplasm was finely granular and stained faintly. The nuclei were rich in chromatin and had distinct nuclear membranes. The cells were slightly larger and vacuolation was more marked and blood-vessels were more abundant than in the normal structure. The capsule was not invaded. Endothelial cells were present and also eosinophil and plasma cells. In places there was but little fibrous tissue present and the cells were in sheets.

Malignant Tumours (*Cases* 1 *and* 2).—The cells in these cases were in masses, without separating fibroblasts. *Case* 2 was obviously the more malignant, not only clinically, but also from the microscopical point of view. Definite invasion of the capsule was present in both cases, but in *Case* 2 the invasion had spread to the surrounding adherent muscle, which also showed a myositis.

The cells were much larger than in the normal organ or in the benign tumours. Vacuolation was gross in *Case* 2, particularly in the centre of the tumour, but the nuclei retained their definite and well-staining properties, and nucleoli were occasionally present. Mitotic figures could be seen. In many places the loose cellular structure had broken down, and hæmorrhages were present. At the periphery of the tumour the vacuolation was less conspicuous and endothelial cells were present in abundance, particularly in those regions where the tumour was invading its capsule and adherent muscle. The blood sinuses were large. It is noteworthy that the number of eosinophil cells was in excess of that normally found in blood sinuses.

Diagnosis.—The definite diagnosis of a fixed malignant carotid body tumour before microscopy would appear impossible. In the past, simple tumours have rarely been given a correct pre-operative diagnosis, mainly because of their rarity. Considering the recorded cases and those mentioned in this paper, the diagnosis of simple or early malignant tumours of the carotid body would depend upon the following characteristics: (1) The tumour is slow-growing. Therefore a large tumour in the neck, of short duration, is unlikely to have arisen from the carotid body, unless there is obvious evidence of malignancy. (2) The situation at the bifurcation of the common carotid artery. (3) Free mobility laterally, but not in the line of the vessels. This lateral mobility diminishes when the tumour is malignant. (4) Practically always unilateral and equally common in both sexes. (5) No fixation to the skin. (6) Transmitted pulsation. (7) Painless and not tender to palpation. (8) The tumour may in rare instances cause syncopal attacks. (9) When the tumour is large, pressure symptoms may occur, e.g. aphonia, dysphagia, dyspnœa, etc. (10) Metastases have not been found, even in malignant cases.

Bevan and McArthy[6] analysed most of the recorded cases. A few of their figures will be quoted: (1) The majority of cases occur between the ages of 30 and 40, and practically 70 per cent between 30 and 60. The average age is 41·7 years. (2) The average duration of the tumour before consultation is $7\frac{1}{6}$ years. (3) Of carotid body tumours 80 per cent are benign.

Treatment.— In considering the treatment of these tumours there are certain facts to be borne in mind: 80 per cent of carotid body tumours are said to be non-malignant; 70 per cent occur between the ages of 30 and 60. The risk attached to resection of portions of the carotid arteries together with the bifurcation of the common vessel is very considerable, and particularly after the age of 40. The mortality is stated to be 30 per cent, and hemiplegic signs occur in some of the patients who survive.[6,7] Therefore, should resection of the vessels be necessary, the operation is fraught with greater danger in the majority of cases. Thus, it would appear that an exploratory operation should be undertaken in all cases clinically operable. If the tumour proves removable without resection of the internal and common carotid arteries, this procedure should be adopted. A tumour which is found to be irremovable except by resection of portions of the vessels mentioned should be left, unless the condition of the patient warrants the severe operative risk. Syncopal attacks definitely due to the tumour, or evidence of malignancy as shown by diminished lateral mobility or microscopy, would be the chief indications for this procedure. The age of the patient, and particularly the condition of the arteries, should also be taken into account.

Radiotherapy has not been used in a sufficiently large number of cases to enable any definite idea to be formed of its value. In one of the malignant cases recorded, radium treatment was followed by no permanent benefit, and Birman[8] observed the same result. Radium should therefore be reserved only for those malignant cases whieh are inoperable, as its beneficial effect is too uncertain.

I am indebted to Sir Percy Sargent and Mr. Max Page for permission to publish these cases, and also to Professor L. S. Dudgeon for much help and advice.

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THE OPERATIVE TREATMENT OF POTT'S PARAPLEGIA.

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My object in writing this paper is to advocate early decompression for Pott's paraplegia in adults. The decompression may take the form of laminectomy, costotransversectomy, or both; but, whatever the method of decompression, I regard stabilization by bone-grafting of the diseased part of the spine as an essential complement.

This paper refers particularly, though not exclusively, to adults, for in my experience children with spinal caries under modern treatment very rarely suffer from severe paraplegia. Probably this is because we are learning to keep them at rest sufficiently long, with the spinal column relieved from strain and metabolism stimulated by open-air conditions. The surgical rest will almost always quickly cure the earlier milder paraplegia, and time, if sufficiently prolonged, prevent the development of late paraplegia—for this disaster has been usually associated with insidious persistent caries after the child had been allowed to get up. On the whole, I would say of Pott's paraplegia that in children it can as a rule be avoided or quickly cured by conservative treatment, but that in adults operation is generally indicated.

There has recently appeared a monograph by Madame Sorrel[1] in which conservative treatment of Pott's paraplegia is advocated. Madame Sorrel does not advocate operation because: (1) In early cases she prefers to continue conservative treatment in the expectation that in the majority the paraplegia will sooner or later disappear; (2) In late cases her experience is that the great majority do not get well whether anything is done or not.

Nevertheless I am at present a whole-hearted believer in operation on patients suffering from severe paraplegia, whether it has appeared early or late. I have long been convinced of the neurological advantage of early operation. Simultaneous bone-grafting has altogether removed the great drawback of laminectomy, and the results have been most encouraging. I feel that the combination of grafting with laminectomy and the stabilization so achieved make a great, and indeed a critical, difference to the arguments for and against decompression. The cure of Pott's disease depends on two things: good general health, and (this is our immediate concern) restored spinal stability. Caries undermines the stability of the spinal column, the resultant instability in its turn favours further erosion, and a very vicious circle is thus set up. Now laminectomy has been discountenanced because it produces a further structural defect, and this is in truth a drawback which only very strong indications could overcome. But if, by combining grafting with laminectomy we can increase, not diminish, the stability of the spine, this drawback exists no longer, and the whole outlook is changed.

I believe that decompression is indicated in many of the early cases in adults, both because some do not clear up at all without operation, and because in many of the others there is a long period of compression which is dangerous and harmful to the cord. Also this long-continued paraplegia is harmful and most distressing to the patient, and this at a time when restoration of his lost vitality is an urgent need. Instead of peace and progress, the patient experiences painful muscular spasms, is with difficulty protected from pressure sores, and, if the sphincters are affected, from ascending genito-urinary infection. Pott described this condition as " most miserable to endure, most pitiable to see ".

As has been said already, in my part of the world Pott's paraplegia in children is now becoming extremely uncommon. I have had hundreds of children suffering from spinal caries under my care, and in the last ten years remember only two or three cases of paraplegia. Indeed, I have only twice had to do a laminectomy for Pott's paraplegia in a child. So what I have to say concerns more particularly Pott's paraplegia in adults.

In my experience paraplegia generally comes on in the first few months of the disease, and in these cases the pressure is almost always from abscess, granulation tissue, débris, etc., in front of the theca. If the paraplegia advances quickly, or if it advances progressively during immobilization, I operate to relieve compression by costotransversectomy or laminectomy, or both. In addition I do a twin graft fixation ; after laminectomy these grafts bridge the opening and more than counteract the local loss of laminæ and spines. In effect a mobile chain of laminæ is replaced by a pair of strong grafts which hold the diseased area of spine much better than any external splint can do. The grafts must be stout, for the strain on them is great ; the stresses are largely concentrated at the level of the disease, and it is here that the grafts are liable to fracture, or to show discontinuity as a result of local osteoclasis from overstrain of the bone-cell-spicule units.

Early decompression is, then, I believe, indicated to protect the cord from prolonged pressure and the patient from the complications of paraplegia ; and grafting, which leads to complete local immobilization of the spine, is indicated to protect the spinal column from further erosion and to promote healing of the lesion.

There are those who think that laminectomy is not helpful in Pott's paraplegia because the pressure almost always comes from in front : they hold that the cord is not compressed against the laminæ but strangulated by being tied down against this anterior pressure by the intervertebral nerves and their dural attachments. This view appears to me theoretically unsound, and it is contradicted by my experience both in operative findings and in the results of laminectomy. *Case* 1 is particularly convincing. Not only was the complete paraplegia very rapidly relieved by laminectomy, but when the paraplegia recurred it proved to be due to the pushing of the theca *backwards through the laminectomy opening* and the compression of the cord against the lower edge of the opening. This was a case of rapidly advancing paraplegia ; and yet the theca not only was first compressed against the laminæ, but after this pressure was relieved by laminectomy the theca was gradually pushed even further backwards and bulged beyond the level of the laminæ

(*see Figs.* 121, 122). In this case without doubt it was the laminæ and not the nervous and dural attachments of the theca which opposed its backward displacement by pathological pressure from in front. This is, I believe, the rule whenever there is gross extrathecal pressure from in front.

Now for a moment to consider spinal caries apart from the paralysis: in 1923 I advocated operative fixation for all adults suffering from spinal caries where no contra-indication existed,[2] and I have learnt to value operative fixation more and more during the seven years since then. Operative spinal fixation in good hands is almost free from risk, and there is little or no disturbance of the lesion if a technique is adopted which maintains immobilization of the spine before, during, and after the operation. I believe that one should graft early, but not until three conditions have been awaited. The patient should be settled comfortably on his frame or plaster bed, and past the period of active ill health; further, the number of vertebræ involved by the disease should be known so that the length and site of the grafts may be correctly judged. Now by the time paraplegia (other than the transient form) has developed, the extent of the disease has become clear. But grafting shuts the door on laminectomy. I have once done a hemilaminectomy on a case in which two grafts had been applied deep down on each side of the spinous processes. This operation was so difficult as to be dangerous, for a thick continuous layer of bone replaced the chain of laminæ. Clearly, then, it is wise in cases of severe paraplegia to do an adequate laminectomy before putting in grafts.

In the last ten years I have had under my care 12 cases of Pott's paraplegia on whom I have operated for relief of pressure on the cord. The operations have been as follows: costotransversectomy and graft 2, laminectomy and graft 5, laminectomy and graft + costotransversectomy 4, laminectomy alone on a case which I had previously grafted 1. In one case where a graft fractured after a fall I re-grafted and at the same time re-opened the costotransversectomy and evacuated a further collection of débris, so that there have been altogether thirteen operations. In most cases the operative interference is completed at one stage, but in three of the cases where a laminectomy, graft, and costotransversectomy have been done, the laminectomy and graft has been at one stage and the costotransversectomy at a second. There have been two deaths, both in late and clearly almost hopeless cases. In one, that of an old lady, death occurred within twenty-four hours from shock. The other case, a man of 34, died about eighteen months later in coma. One case of laminectomy, graft, and costotransversectomy has been done too recently to allow of any progress report other than that the patient was undisturbed by the operation. Of the other 9 cases, recovery, partial (4) or complete (5), has occurred. It is noteworthy as regards two of the cases with complete recovery that the paraplegia had occurred late on in the second year of the disease. Further, of the four partial recoveries, two are still improving progressively and seem likely to recover completely.

There are, then, three operations to be considered: (1) Costotransversectomy; (2) Laminectomy; (3) Grafting. But even if all three are done (in one-third of my cases) there need only be two stages, for the grafting is a supplementary part of the laminectomy operation. The first question is

whether to do a costotransversectomy or a laminectomy. If this doubt arises, it is often wise to do both; then it remains to decide which should be done first. The argument in favour of doing the laminectomy first is the advantage of the earlier immobilization following the graft, for in Pott's disease with paraplegia one is especially anxious to limit the local destruction and the piling up of the 'granuloma-débris dump' as quickly and decisively as possible. On the other hand, costotransversectomy may eliminate the need for laminectomy. But in considering this one should remember the occasional occurrence of a tuberculous sinus after a costotransversectomy through tuberculous infection of the wound track. (*See Case* 6, in which a sinus developed four to five weeks after operation.) Fortunately such a sinus takes several weeks to appear, so there is time during which one can generally get guidance as to the need for laminectomy, and then carry it out as part of the graft operation after the costotransversectomy and before the time when a sinus might develop. Such a sinus, though uninfected by pyogenic organisms and several inches away, would make one much less happy in performing the laminectomy–graft operation.

I favour doing the costotransversectomy first if X rays show a spherical, or fusiform but nearly spherical, prevertebral abscess, for this appearance suggests that there is still a tight and localized distension of the prevertebral fascia by débris which, unable to escape upwards or downwards, has found its way backwards. In such cases I generally do a costotransversectomy first, then a fortnight or so later a graft, and unless the paraplegia is improving I do a laminectomy also at this second operation. It is occasionally advisable to evacuate the débris a second time if the clinical state and radiograms indicate the need (*see Cases* 6 and 9).

Then with regard to the second group of cases in which paraplegia comes on late in the history of Pott's disease, Madame Sorrel's view is that nothing will cure 90 per cent of these, and therefore she counsels inaction. Admittedly these late cases are much less hopeful. They are less likely to get over the tuberculous disease, and the local cause of the compression is less often uncomplicated extrathecal compression. It appears to be impossible at present to pick out with any certainty those patients whom operation will relieve or cure; probably it is still harder to distinguish those in whom prolonged compression has led to permanent changes in the cord or in whom the paraplegia is due to widespread thickening of the dura. My own view is that one should frankly explain the situation, then advise operation, but only perform it if the patient desires it, being fully conscious of the uncertainty of the outcome. I would advise operation even for those who from age or lack of vitality seem little likely ultimately to recover from the tuberculous disease, for I would myself so far prefer a recovery from paralysis, a period of symbiosis with tubercle bacilli, and the milder death of generalized tuberculosis, to continued paraplegia with all its discomforts and an end no less inevitable and probably preceded by sores and urinary sepsis. But I am much more hopeful than Madame Sorrel as to the percentage of late cases which can be cured or relieved. *Case* 8 shows that operation, even in a very late case, may be worth while. Fortunately I see few of these late paraplegias, probably because I work in a district in which almost all the cases

of spinal caries come early to the orthopædic hospital and get prolonged general treatment and careful after-care. Also almost all the adults are grafted as soon as the indications are satisfied, and I feel very strongly that in adults the strong double graft which I use is a very effective internal splint, which checks erosion and débris production, limits the extension of the disease, and thus minimizes the chances of paraplegia.

CASE REPORTS.

Case 1.—Male, age 19. Admitted to the Wingfield Orthopædic Hospital on Jan. 12, 1921.

HISTORY.—Kyphosis in dorsal region present for an indefinite period. Onset of loss of power in both legs commencing three weeks before admission. Patient walked up to out-patients' department with spastic gait.

X RAY.—Bodies of D.6, 7, and 8 largely destroyed. Abscess shadow.

Jan. 12, 1921.—Voluntary power: right and left legs nil. Spasms: right and left legs + +. Sensation: light touch lost below 8th dorsal. Sphincters: not

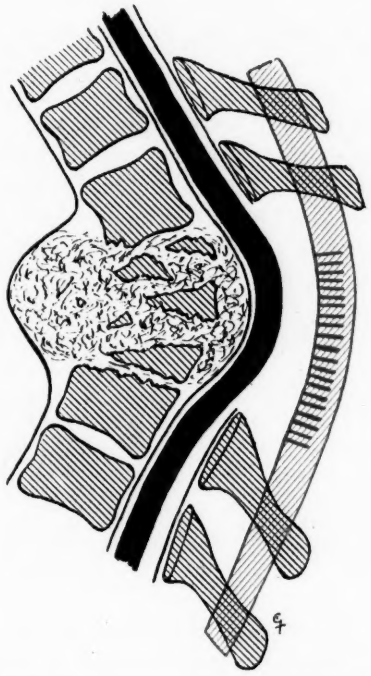

FIG. 121.—*Case* 1. Condition at the end of the first operation (Jan. 28, 1921). Graft shown in red, with saw cuts to allow flexion.

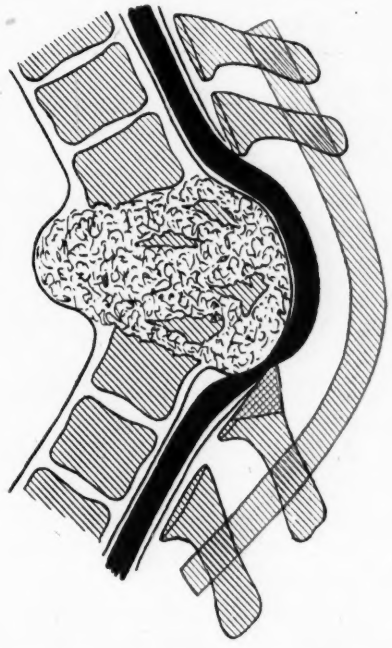

FIG. 122.—*Case* 1. Compression found at second operation (June 12, 1921). Area of further laminectomy shown cross-hatched in red.

affected. Reflexes: abdominals −; knee-jerks + + +; ankle-jerks + + +; clonus, present; plantar, extensor (all right and left). Wassermann: negative.

Despite immobilization on spinal frame the paralysis deepened, and became complete, motor, sensory, and sphincter.

Jan. 28, 1921.—*Laminectomy and graft.* D.6, 7, and 8 spines and laminæ removed. Dura pushed through opening at once and began to pulsate well. Prominence palpable in front of cord. This had clearly compressed the cord against the laminæ. Tibial graft fitted to bridge gap from two spines above to two

spines below. Many transverse cuts to make it fit. Immediate result: Sensation and muscular control of the lower limbs recovered the same evening; but this improvement was not maintained, and in two or three weeks there was complete sensory and motor paralysis and incontinence.

June 12.—*Enlargement downwards of laminectomy opening* without damage to graft. Graft and membranes inspected. The theca was found projecting backwards through the laminectomy opening and pushed against the lamina at the lower edge of this opening. I found it possible to remove enough of this lamina by undercutting the graft.

Fig. 121 indicates the state of affairs at the end of the first decompression. *Fig.* 122 shows the compression found at the second operation owing to increased accumulation in front of the theca. It was noteworthy that the graft was rounded, covered with a vascular fibrous membrane, and had retained its arch; it appeared to form an extremely strong and fully adequate internal splint.

July 14.—Incontinence of urine and fæces ceased.

Dec. 30.—Complete recovery of sensation and sphincter control; partial of voluntary power.

Dec. 12, 1922.—Getting up in spinal support and calipers and learning to walk.

March 21, 1923.—Great improvement in walking.

April 30.—Discharged.

March 3, 1926.—Very well indeed.

This patient is now perfectly well and active. There is no trace of paraplegia left. He has been at work for years.

Comment.—This was a case in which the relief of paraplegia was dramatic in its swiftness and completeness. The onset of paralysis was rapid and total, and the recovery of sensation and movement in the lower limbs followed operation immediately. A relapse followed owing to enlargement of the mass in front, and an insufficient area of decompression. Relief was again obtained, though not so swiftly, by a second operation and removal of more bone at the lower angle of the laminectomy opening. The final result has been complete restoration of function and a normal working life.

There are several points of great interest in this case:—

1. It was the loss of power and awkwardness in his legs that brought the patient to hospital.

2. The loss was rapidly progressive from the time when the patient was first seen until the date of operation, although he was kept completely lying down until he could be admitted and immobilized on a spinal frame.

3. Laminectomy was done early, and the relief of paralysis was complete and immediate.

4. But it recurred, and, as was found at the second operation, was due to further accumulation of débris between the centra and the theca and to crushing of the theca against the lamina as shown in *Fig.* 122.

5. The second operation was not done early, and the recovery was delayed, though ultimately complete.

6. I would add that nowadays I should have done a costotransversectomy before or within a week or two of the laminectomy and graft, and I feel sure that this would have prevented the return of the paralysis.

It was difficult to decide whether 'to go in again' early or not, and I am confident that a quicker recovery would have followed either costotransversectomy and evacuation of the abscess or an earlier extension of the laminectomy.

The second operation demonstrated the possibility of reaching the theca to one side of a graft bridging a laminectomy. This is made easier when the graft or grafts are not bedded deeply. It also enabled me to see how very strong the graft was—a thick rounded living bridge, covering, protecting, and completely immobilizing the area of disease and laminectomy.

Case No. 2.—Male, age 26. Admitted to the Wingfield Orthopædic Hospital on Jan. 24, 1921.

HISTORY.—Injury to back in 1918. Pain in back ever since; worse since July, 1920. Pain in abdomen in August, 1920. Numbness of legs and later loss of power in September, 1920.

X RAY.—Shows considerable destruction of bodies of D.8 and 9, with abscess shadow.

Voluntary power: right and left legs, nil. Spasm: right and left legs ++. Sensation: light touch +; pinprick diminished below 8th dorsal segment. Incontinence: difficulting in starting and scarcely aware of act for both urine and fæces. Reflexes: knee-jerks ++; ankle-jerks +; clonus (ankle) +; plantar, extensor (all right and left). General condition very poor.

March 14, 1921.—*Laminectomy.* Laminæ of D.7, 8, and 9 removed. Pulsation absent till D.7 laminæ removed, then returned at once. Carious material found and removed from posterior surface of body of D.8, which appeared widely destroyed, and showed considerable respiratory movement. In view of poor general condition, graft was postponed.

May 27.—All muscles acting strongly in right and left legs. Very few spasms.

Oct. 10.—Still complaining of pain across chest and to back. (This persisted until graft.)

Nov. 21.—*Graft.* Tibial graft from D.4 to D.9 inclusive, bridging laminectomy gap. Multiple transverse cuts to adapt graft to kyphos.

August, 1922 (approx.).—Discharged wearing spinal support.

N.B.—Discharged unduly early as he could not stay in hospital owing to very bad home conditions of wife and family.

Feb. 25–Oct. 22, 1925.—Re-admitted and put on frame for pain in back and chest and general ill health. Very bad home conditions. No paraplegic symptoms or signs.

X RAYS.—Showed discontinuity of graft opposite D.9 and abscess shadow to right of D. 8, 9, and 10. Discharged better and wearing support.

N.B.—In my present practice I should have patched and reinforced the graft.

Feb. 14, 1929.—Re-admitted for pain in back and chest, weakness in legs, and general ill health. No definite clinical signs of paraplegia.

X RAY.—Discontinuity of graft, and abscess shadow.

March 5.—Definite paraplegic signs and tendency to incontinence. On plaster bed for operation.

April 22.—*Operation.* (1) Two fresh tibial grafts cut and laid alongside old ones on each side over whole length of area; (2) Costotransversectomy on right side begun, but abandoned incomplete owing to condition of patient.

May 20.—No improvement. Voluntary power: right and left legs, nil. Spasm: right and left legs +++. Sensation: below 8th segmental level −. Incontinence: automatic bladder and rectal action with no voluntary control. Reflexes: knee-jerks + + +; ankle-jerks +; plantar, extensor; clonus (ankle) +; abdominals − (all right and left).

May 21.—*Costotransversectomy.* Vertebral end of rib and transverse process of corresponding vertebra opposite kyphos on right side excised and much bone sand curetted away. No large sequestra. No pus.

Feb. 17, 1930.—Soundly healed. General condition excellent. Voluntary power: right and left legs, complete. Spasms: right and left legs, nil. Sensation: diminished (but present) below 8th dorsal segmental area. Incontinence: nil. Reflexes: abdominals −; knee-jerks + +; ankle-jerks + +; clonus (tendency, right), left, nil; plantar, extensor (all right and left).

Comment.—Here the onset of paralysis was gradual, but had become practically total when the patient was admitted to hospital some five months after the first appearance of symptoms. Laminectomy and later graft resulted in restoration of function sufficient for discharge in seventeen months. His home circumstances were very difficult, and the strain to which he was unavoidably exposed led to recrudescence of activity in his spinal focus. Finally paraplegia recurred eight years after his original laminectomy. For this he was first re-grafted, and as this with its necessary accompaniment of complete immobilization produced no improvement in his condition, costo-transversectomy was performed and much tuberculous material thereby given an exit. This has resulted, nine months later, in practically complete restoration of function, the only residual signs now being a slight impairment of sensation and increased reflex activity in the lower limbs.

Case 3.—Male, age 34. Admitted to Wingfield Orthopædic Hospital on Dec. 14, 1922.

HISTORY.—Pain in back and along costal margin for eighteen months. Kyphos over D.9 and 10. Wassermann, negative.

X RAY.—Destruction of bodies of D.9 and 10.

April 20, 1923.—*Graft.* Single tibial graft applied, long and broad, on left side.

Sept. 7, 1926.—*Re-graft.* Re-grafted from D.7 to 11 on right side, owing to disease being shown above original graft.

Dec. 17.—Spinal support applied.

March 29, 1927.—Discharged home.

Sept. 12.—Re-admitted, complaining of pain in abdomen and across back since May 1.

Dec. 8.—*Drags legs* as he walks. Right leg weak. Sensation: ? a little dull in right and left legs. Reflexes: knee-jerks + +; ankle-jerks + +; clonus (ankle) +; plantar, left extensor, right ? extensor (all others right and left).

Dec. 22.—Some difficulty in beginning micturition.

April 29, 1928.—Back on frame.

May 7.—Voluntary power: practically nil; can dorsiflex toes slightly. Spasm: +. Reflexes: knee-jerks + +; clonus (ankle) +; plantar, extensor (all right and left); abdominals + + above umbilicus, − below. Incontinence: difficulty in starting better since on frame.

May 10.—*Laminectomy.* Spines of D.9, 10, and 11 exposed. Graft most solidly fused to spines, cut away with difficulty with motor-saw, and preserved for re-insertion. Laminæ of D.10 removed. No pulsation of dura. Probe passed downward freely, but not upward. Laminæ of D.9 removed. Probe passed again upward tapped or ruptured a ? extradural cyst, and liberated a small quantity of turbid fluid. Flow did not continue, therefore not cerebrospinal fluid. Pulsation immediately started. A little more bone then removed in upward direction, and the old graft replaced, bridging gap. Operation followed by retention of urine and paralysis of both lower limbs of flaccid type, with partial loss of sensation in right leg and complete in left leg.

May 21.—Voluntary power: right and left legs, nil. Spasm: right and left legs, nil. Pinprick sensation: right leg, impaired; left leg, absent. Incontinence: rectal, complete; bladder, drained by catheter tied in and changed twice weekly. Reflexes: knee-jerks +; ankle-jerks +; clonus −; plantar, flexor (all right and left); abdominals upper abdomen +, lower abdomen −.

May 31.—*Return of spasticity.* Voluntary power: right and left legs, nil. Spasm: right and left legs +. Sensation: as on May 21. Incontinence: as on May 21. Reflexes: knee-jerks +; ankle-jerks + +; plantar extensor (all right and left); abdominals as on May 21.

June 30.—Automatic action of bladder established. No voluntary control.

Aug. 4.—Both lower limbs definitely spastic with increased reflexes; extensor,

plantar. Moving big toes and constant spasms. Complete anæsthesia and paralysis in left leg. Impaired sensation and slight power to move foot and toes in right leg. Bladder and rectum: automatic. No voluntary control.

Nov. 18.—Able to raise right leg off frame. No other change.

Jan. 22, 1929.—Discharged home at own request. During last four months the patient had curious periodical attacks of pyrexia lasting six to seven days, the temperature rising to a peak of 103° or so, and accompanied by severe headache and sweating, but no rigors. No cystitis was present. No important change in neurological state occurred. No recovery whatever of voluntary power or sensation in the left leg. Was able to raise the right leg and move the foot and toes, and sensation in this limb became practically normal. Both legs remained spastic, with spasms and increased reflexes and upward-moving big toes. Bladder and rectum: automatic, with no voluntary control.

Comment.—This is a case of late paralysis, onset five years after original admission. In addition the previous double grafting made laminectomy very difficult. This man died eighteen months later in coma.

Case 4.—Male, age 21.

HISTORY.—Pain and stiffness in back for six weeks, then fifteen days previously sudden onset of acute pain in back followed a few hours later by numbness and paralysis of legs. He was then admitted to the Radcliffe Infirmary, Oxford, and transferred to Wingfield Orthopædic Hospital on Oct. 29, 1923.

Voluntary power: right and left legs, nil. Spasms; right and left legs, nil. Sensation: complete loss below line posterior inferior spine, anterior superior spine, middle Poupart's ligament, symphysis pubis, right and left. No zone of hyperæsthesia. Reflexes: knee-jerks − ; ankle-jerks − ; clonus, nil; plantar, nil (all right and left); abdominals, upper +, lower −. Incontinence of bladder and rectum.

Summary. — Complete flaccid paralysis of both lower limbs, with loss of sensation and incontinence.

FIG. 123.—*Case* 4. Skiagram showing bellows-like collapse of the vertebral body, with upper and lower discs little affected.

Oct. 30, 1923.—*Laminectomy and graft.* Laminæ of D.10, 11, and 12 removed. Pulsation free above, none in lower quarter of exposed area. After full exposure flattened area opposite D.11 filled out, but pulsation never seemed as full as at higher level. There was a prominence of the theca opposite D.11. Aneurysm needle passed in front of theca at this level failed to find débris. Double lateral tibial graft from D.8 to upper lumbar, bridging laminectomy gap.

April 28, 1924.—Nephrectomy (right) for pyelonephritis, with removal of ureter containing stone.

April 15, 1925.—Voluntary power: left leg greatly improved, right leg not so good. Spasms: left + + (after effort), right + + with very slight provocation. Sensation: no tactile sensation below either knee. Incontinence: complete.

Aug. 31.—Walking with stick and one person's help. Has to wear urinal.

Oct. 30.—X rays show the collapsed vertebra with the intervertebral space above normal and below not obliterated—grafts in position (*Figs.* 123, 124).

Dec. 16, 1927.—Still incontinent and spastic.

Feb. 29, 1928.—Walking fairly well.

Feb. 19, 1930.—Walks with the help of two sticks quite well, though with stiff spastic type of gait. Voluntary power: practically normal in right and left legs; dorsiflexion of feet incomplete owing to spasm of calf flexors. Spasm: right and left legs + (after exertion). Sensation: diminished though present below same line as before operation (right and left). Incontinence: voluntary control of bladder and rectum still not complete. Reflexes: knee-jerks + + +; ankle-jerks + + +; plantar, extensor; clonus (ankle and patella) +; abdominals + + (all right and left).

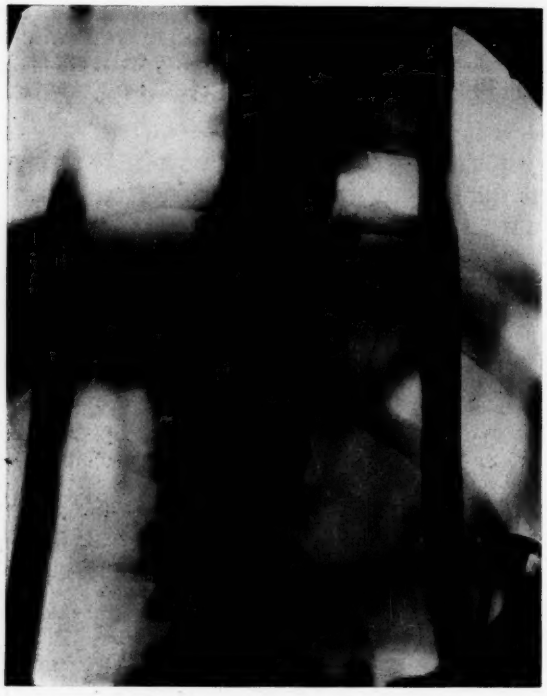

FIG. 124.—*Case* 4. Skiagram after laminectomy and grafts.

Comment.—Apparently the 11th dorsal vertebral body collapsed suddenly, and, shutting up like a pair of bellows, shot its semi-fluid contents backwards, causing so sudden a compression as to produce a traumatic flaccid paralysis. The radiogram (*Fig.* 123) shows the upper and lower discs of D.11 in contact. At the laminectomy it was clear that the theca was still compressed between the prominence in front and the laminæ.

Laminectomy and graft was followed by considerable improvement, the paraplegia changing to the spastic type, and voluntary power returning sufficiently to permit of walking with a stick; but the patient has never recovered sufficient activity to resume working life. He still walks with a stick with a stiff spastic gait, is slightly incontinent of urine and fæces, and is liable to occasional spasms.

Case 5.—Male, age ? 35. Admitted to Wingfield Orthopædic Hospital on Oct. 17, 1923.

HISTORY.—Old tuberculous hip, and had three months' sanatorium treatment in 1917 for hæmoptysis; no tubercle bacilli found in sputum. Pain in back in March, 1923, followed by development of kyphos. Kyphos D.8 to 12.

X RAY.—Shows destruction of bodies of D.9 and 10, with collapse and lateral displacement.

Motor power: very spastic. Spasms: + + (right more than left leg). Sensation: right leg impaired. Incontinence: bladder distension, impairment of rectal sphincter control. Reflexes: knee-jerks + + (right more than left); clonus (ankle and patella) + (right more than left).

Nov. 22, 1923.—*Laminectomy and graft.* Three spines opposite kyphos, together with two above and two below, exposed. Laminæ of three vertebræ removed. Dura

pulsated freely in upper part. No pulsation below middle of exposed area. The vertebral canal was much narrowed and distorted by double lateral curve so that the theca was confined and had been compressed over a distance of 2 to 3 in. After removal of laminæ the compressed area filled out, but pulsation did not return to the middle and lower portion. Ridges of bony opening were smoothed and two tibial grafts inserted, bridging the laminectomy gap from two spines above to two below.

July 7, 1924.—Spasms much less. Improving greatly.

Oct. 1.—Off frame. Spinal support applied.

April 15, 1925.—Great improvement. Is wearing back splint at night to prevent contracture. Balance still poor. Legs still very spastic. Plantar response, flexor. Almost complete absence of sensation below knees. Walks with two sticks.

Feb. 1, 1926.—Re-admitted for pain over graft area. X-ray appearances suggested fractures of the grafts.

Feb. 8.—*Operation.* Grafted area exposed and inspected. No solution of continuity in grafts, which appeared firm and sound. Fusiform thickening of bone in centre of area possibly due to old fracture. Wound closed without further interference.

March 3.—Has resumed back support. No pain in back.

Oct. 30, 1927.—Admitted to Abingdon Sanatorium for some return of adduction spasm in legs. X rays showed solution of continuity of graft opposite point of maximum tension, but without any separation. Much granulation tissue round spine. Costotransversectomy considered.

July 9, 1929.—No pain in back. Less spasm in legs. Looking well.

Feb. 24, 1930.—Walks fairly well, but with dragging spastic gait. Voluntary power: practically normal. Spasms: + occasionally. Reflexes: knee-jerks + +; ankle-jerks + +; clonus +; plantar extensor (all right and left). Incontinence: still some weakness of bladder control.

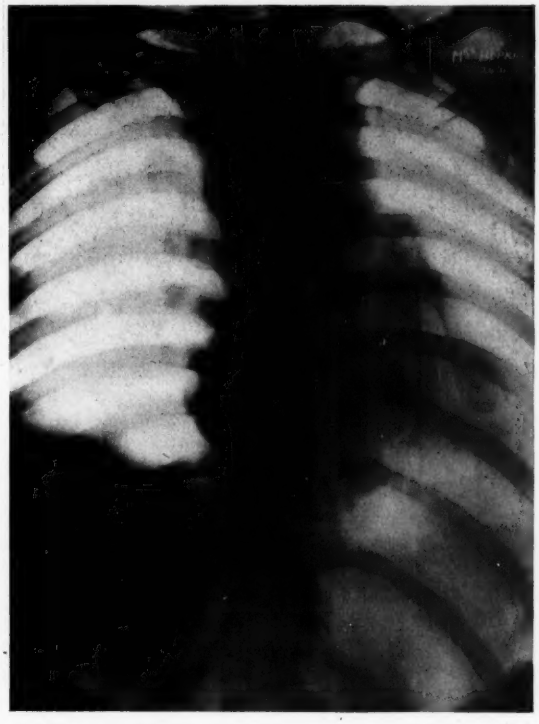

FIG. 125.—*Case* 6. Skiagram showing large tense abscess in site of previous costotransversectomy.

Comment.—In this case also the onset was gradual and was well marked, though not complete, at the end of six months, the symptoms and signs here involving one limb more severely than the other. The laminectomy and graft was successful in relieving the paraplegia, but in sixteen months' time the patient was walking with two sticks and still spastic. He is able to get about with sticks and no pain, and has only minor discomfort from occasional muscular spasm and a slight persistent incontinence of urine. This and the persistent spasticity of his lower limbs debar him from leading an active working life.

Case 6.—Female, age 33. Admitted to Wingfield Orthopædic Hospital on July 10, 1924.

HISTORY.—Previous history of tuberculous glands and pleurisy. Six months' history of pain and weakness in back. Tender over small kyphos in mid-dorsal region.

X RAY.—Caries of body of D.8, angular deformity, and abscess shadow.

Central nervous system: legs spastic; knee-jerks + +; plantar, flexor. Signs of paraplegia increased. Spasms and tingling of legs became troublesome.

Oct. 28, 1924.—*Costotransversectomy* 9th right rib. Large abscess evacuated.

Oct. 31.—Now no spasms.

Nov. 11.—*Albee graft.*

Aug. 2, 1925.—Discharged.

Nov. 23.—Re-admitted after a fall leading to fracture of graft.

Nov. 24.—X rays show shadow of large tense abscess (*Fig.* 125).

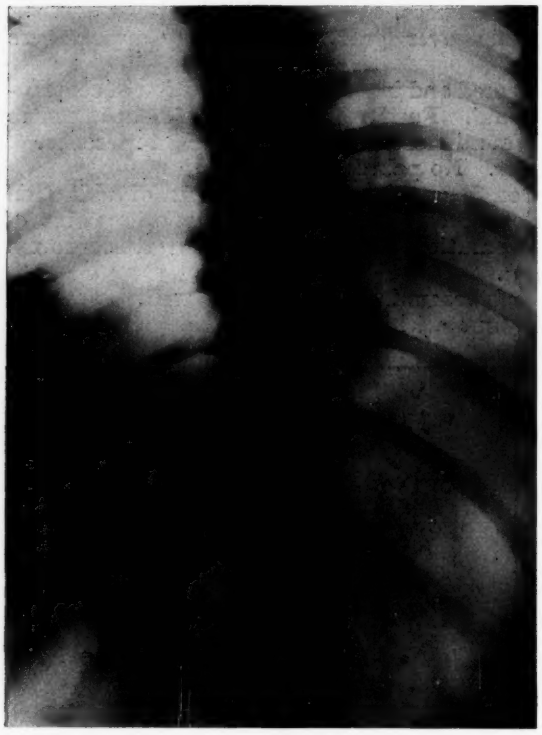

FIG. 126.—*Case* 6. Skiagram showing disappearance of the abscess after reopening of the costotransversectomy.

Dec. 15.—*Repair of graft.* Linear transverse fracture found about middle of graft. New 4 by ¼ in. graft inserted into bed in old graft. This was made by a long vertical motor-saw cut to the left of the centre of the old graft so that with the main part of the graft left undisturbed the left side of it could be mobilized outwards.

Re-opening of costotransversectomy. About an ounce of pus and three small sequestra with bone sand removed. Toilet of wound with ether. Wound closed.

Dec. 22.—Turned. Sutures removed. Wounds well healed.

Jan. 23, 1926.—Turned. Wound corresponding to costotransversectomy broken down at area size of sixpence about its middle. General condition good.

Feb. 19.—Turned. Clear fluid, one or two drops only, from sinus, otherwise satisfactory. General condition very good.

March 16.—Off frame. Spinal support satisfactory.

April 5.—X rays show disappearance of the large abscess shadow (*Fig.* 126).

April 30.—Discharged. Still small sinus.

May 14.—Seen at home. Doing very well.

June 8, 1928.—Sinus healed. General condition good. To continue support.

Sept. 14.—General condition good. Leaving off support with no ill effects.

March 14, 1930.—Very well. Leading normal active life. Voluntary power: normal. Spasm: nil. Sensation: normal. Reflexes: knee-jerks + +; ankle-jerks +; plantar, not obtained; clonus −; abdominals + (all right and left). Incontinence: nil.

Comment.—In this case also there was gradual onset of paraplegic symptoms which never became complete. Costotransversectomy led to evacuation of a large abscess and resulted in disappearance of the pain and muscular spasms. A tibial graft was subsequently inserted, which fractured a year later as the result of a fall and necessitated a further operation for repair of the graft. At the same time the prevertebral abscess was again evacuated. Result: paraplegia cured.

Case 7.—Female, age 56. Admitted to Wingfield Orthopædic Hospital on Aug. 30, 1926, from Royal Berkshire Hospital.

HISTORY.—Ten weeks' pain in back. Gradual loss of power in legs now complete.

X RAY.—Much destruction of D.10. Spine: kyphosis involving D.9, 10, 11.

General condition extremely poor. Œdema of legs. Flaccid paralysis. Right knee-jerk, just obtainable. Foot-drop.

Later.—Knee-jerk +; ankle-jerk +; ankle clonus +; Babinski present (all right and left). Feet œdematous. No impairment of sensation.

May 12, 1927.—*Laminectomy and graft.* Theca not pulsating, pressed on backwards by sharp edge of bone in front. Collapsed a few hours after operation. Death.

Comment.—Gradual onset of paralysis becoming complete, at first flaccid, later becoming spastic in type. Laminectomy and graft was done, but the patient collapsed and died a few hours after operation. This patient was 56, very feeble, with œdematous legs, and operation was undertaken as a forlorn hope and only as the patient was going downhill with distress and great discomfort.

Case 8.—Male, age 33. Admitted to Wingfield Orthopædic Hospital on April 16, 1929.

HISTORY.—Pensioned from Army in June, 1928, with tuberculosis of the spine. Continued to get pain in back at intervals and was treated in University College Hospital and at Oswestry. Leather support fitted at latter.

In March, 1929, had 'influenza', and on getting about again noticed *weakness and numbness of legs, and difficulty in walking.*

X RAY.—Shows abscess formation at side of extensive caries involving D.10, 11, 12, and L.1.

April 16, 1929.—Voluntary power: gait spastic. Spasms: very slight and occasional. Sensation: normal. Reflexes: abdominals +; knee-jerks + + +; ankle-jerks +; plantar, extensor; clonus, nil (all right and left). Incontinence: nil.

May 21.—*Costotransversectomy.* Left side opposite centre of diseased area. Tuberculous granulomatous tissue and bone sand removed. Wound sutured.

June 21.—*Graft.* Three autogenous tibial grafts used and laid into bed prepared as follows: Two angled ones on right side slightly overlapping at the convexity of the kyphotic curve; one straight one on left side.

Sept. 13.—Off frame. Spinal support applied.

Oct. 13.—Began walking.

Nov. 21.—Last seen as out-patient. Voluntary power: no loss. Spasms: nil. Sensation: normal. Reflexes: abdominals +; knee-jerks + +; ankle-jerks +; plantar, not obtained; clonus, nil (all right and left). Incontinence: nil.

Comment.—This is a case of paraplegia appearing late, and, according to Madame Sorrel, of the incurable type. The paraplegic signs were motor only. They cleared up completely after costotransversectomy followed by grafting one month later. The man is leading an active life.

Case 9.—Male, age 6. Admitted to Wingfield Orthopædic Hospital on Dec. 20, 1928.

HISTORY.—Admitted to Radcliffe Infirmary on Dec. 8. History of ? tuberculous glands removed from neck a year previously. Weakness of legs and incontinence.

X RAY.—Shows collapse of D.6 and 7, with abscess shadows.

Dec. 20, 1928.—No deformity. Slight paraplegia. Some urinary incontinence. X rays show disease of D.6 and 7 (*Fig.* 127).

June 19, 1929.—Voluntary power: nil. Spasm: +. Sensation: normal. Reflexes: knee-jerks + +; clonus (ankle) + +, patella, occasional; abdominals −;

plantar, extensor (all right and left). Incontinence: automatic action of bladder and rectum with no voluntary control.

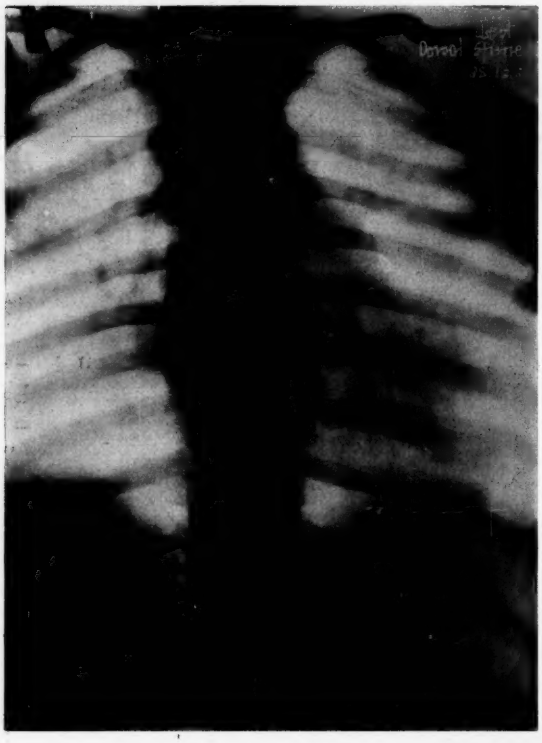

FIG. 127.—*Case* 9. Skiagram showing small abscess shadow.

July 22, 1929.—*Laminectomy and graft.* Laminæ of D.6 and 7 removed. Dura bulged backward, but probe passed easily up and down and laterally. No pus or granulomatous tissue seen. Two grafts cut from left tibia and placed bridging laminectomy gap from D.4 to D.9, inclusive.

Feb. 7, 1930.—Paraplegia persists unchanged. The abscess shadow suggested tenseness, so costotransversectomy was decided upon.

Feb. 10.—*Costotransversectomy.* Grafted area inspected first through old incision. Grafts firm and solid, so not disturbed. Wound closed. Through separate incision vertebral end of 7th rib on left side resected. This was sufficient to liberate tuberculous pus and granulomatous tissue without removal of corresponding transverse process. As much débris as possible removed with spoon, and wound sutured with rubber tube drainage for twenty-four hours only.

Feb. 20.—Wound healed. There has been less incontinence since the operation. Neurological state in other respects unchanged.

July 17.—There is now commencing recovery of voluntary power in the muscles of the thigh on both sides.

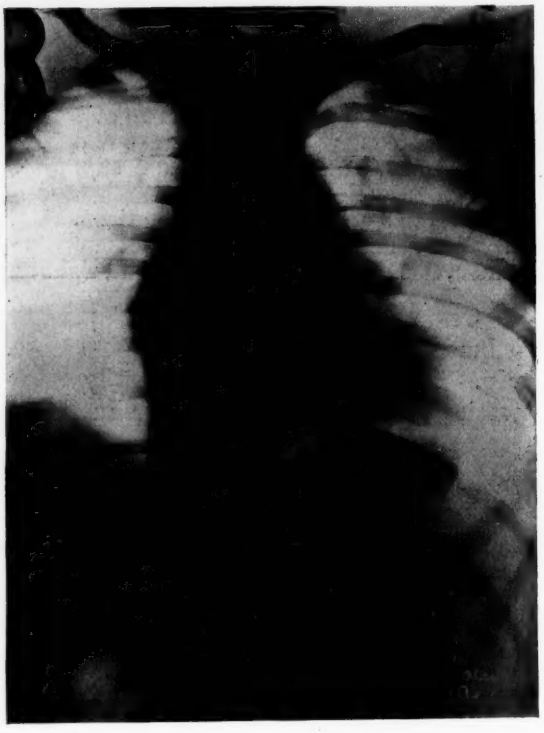

FIG. 128.—*Case* 9. Skiagram taken eight months after a large abscess had been emptied by costotransversectomy. Further accumulation of débris is shown.

Oct. 10.—X rays show further increase of abscess (*Fig.* 128).

Later.—Voluntary power, sphincters, and sensation returned. Still some partial incontinence.

Comment.—A case of paraplegia of early onset, and in a

child, which increased during treatment and showed no sign of improvement after six months' immobilization. Laminectomy and costotransversectomy have led to a delayed but complete motor recovery. There is still incontinence. Presumably earlier operation would have had quicker and better results.

Further, the radiogram dated Oct. 10, 1930 (*Fig.* 128), shows a large abscess shadow. There has been a further accumulation of débris, indicating that it may be advisable to empty the abscess again through the costotransversectomy wound. Clearly general treatment and immobilization were not enough. In such a case one must interfere to relieve the cord from pressure, and be prepared to do so again if the signs and symptoms (always including radiograms) indicate the need.

Case 10.—Female, age 26. Admitted to Wingfield Orthopædic Hospital on Aug. 29, 1929.

History.—Confined in May, 1929. Since then has had pain in left thigh. Recently has noticed a lump in her back and has had difficulty in walking.

Fig. 129.—*Case* 10. Skiagram showing large tense abscess.

Fig. 130.—*Case* 10. Skiagram taken after laminectomy, grafting, and (a week later) costotransversectomy. The grafts and disappearance of the abscess are shown.

X Ray.—Disease D.11 and 12. Angular kyphos with lateral collapse and large abscess shadow (*Fig.* 129). Early paraplegia.

Voluntary power: both legs impaired. Ankle clonus present.

During the next six weeks the paraplegia deepened. Voluntary power: both legs, nil. Spasms: + + (right more than left). Reflexes: knee-jerks and ankle-jerks normal (right and left); clonus, left +, right + + + ; plantar, extensor.

Sensation: anæsthesia to light touch over both heels, and impaired sensation on dorsum of right foot. Inability to distinguish pinprick over corresponding area. Trophic condition round feet: ankles very œdematous, and pressure-sores over anæsthetic area. Sphincters: normal.

Oct. 15, 1929.—*Laminectomy and graft.*

Oct. 22.—*Costotransversectomy*, 11th L., thus opening large abscess and evacuating much caseous material and sequestra. Sufficiently good exposure without removing transverse process.

Feb. 8, 1930.—X rays show disappearance of abscess shadow (*Fig.* 130).

Feb. 22.—Wound soundly healed; graft firm; no paraplegia.

Voluntary power: still slight weakness in right anterior tibial group, otherwise restored. Spasm: nil. Reflexes: knee- and ankle-jerks, normal; clonus, −; plantar, indeterminate. Sensation: no sores; no anæsthesia or hyperæsthesia.

March 21.—All muscles have now recovered and are functioning well, with the exception of extensor proprius hallucis.

May 4.—Back satisfactory; walking well. Some recovery of extensor proprius hallucis.

Comment.—Rather gradual onset, becoming complete in about two months. Laminectomy and graft was followed in a week's time by costotransversectomy in this case. The result has been almost complete recovery from paraplegia as early as four months after operation.

Case 11.—Male, age 34. Admitted to Wingfield Orthopædic Hospital on Feb. 1, 1930.

History.—Three to four months before admission noticed weakness of both legs and dragging of right foot. Unable to walk by end of December, 1929, and then first noticed difficulty with micturition followed later by incontinence. Incontinence of fæces about a month before admission. Complete loss of power in legs and spasms since December.

X Ray.—Disease involving D.3, 4, and 5. Abscess shadow on left side.

Feb. 1, 1930.—Definite kyphos at level of D.3. Voluntary power: nil. Spasms: +. Reflexes: knee-jerks +; ankle-jerks +; plantar, extensor (moving big toe); clonus −; abdominals − (all right and left). Sensation: impaired, but not lost to pinprick below line at level of 6th costal cartilage. Incontinence: automatic action of bladder and rectum, with no voluntary control.

Feb. 10.—*Laminectomy and graft.* Laminæ of D.3, 4, 5 removed. Dura bulged backward and did not pulsate. Probe passed easily upwards and downwards. More bone nibbled away on left side, and the probe passed round, when a small quantity of caseous débris appeared and was removed. Pulsation was not observed to return. Two strong tibial grafts inserted on each side, bridging laminectomy gap from two spines above to two below.

Feb. 20.—Sensation is now normal except for a narrow band of anæsthesia below line at level of 7th costal cartilage. No return of voluntary power or sphincter control. No change in reflexes.

June 27.—Can definitely move voluntarily the outer toes on his right foot, and something in the outer part of the thigh. The effort initiates violent spasms.

Spasms: severe at times.

Sensation: patient finds it very difficult to define his sensory reaction at all clearly. Undoubtedly he can appreciate pinprick everywhere over his lower extremities, though in patches he describes it as 'dull', and in places—especially the soles of the feet—as 'very sharp'. He is not consistent in these localizations, and in fact the reactions do seem to vary. There is a pretty constant line of hyperæsthesia about an inch above the umbilicus.

Incontinence: knows definitely when bladder or rectum is full, and has regained a measure of control over sphincter, though this varies from day to day.

Reflexes: abdominals −; knee-jerks + +; ankle-jerks + + (right and left); clonus (ankle), right −, left +; plantar, right flexor, left extensor. Examination always starts severe spasm.

July 11.—Commencing recovery in muscles of thighs and legs.

Oct. 31.—Further improvement in voluntary power. Spasms still present. Sphincters still only partially under control.

Comment.—Gradual onset of paraplegia, taking three to four months to become complete. Laminectomy and graft has resulted in restoration of sensation to practically normal, and there is commencing return of voluntary power in the lower limbs and of control of bladder and rectum.

Case 12.—Girl, age 15½ years.

HISTORY.—In November, 1922, girdle pains associated with gradual loss of weight. In September, 1923, dorsal kyphos discovered. Treated in supine position till in June, 1926, she was pronounced cured and allowed up in celluloid jacket. In October, 1926, there was X-ray evidence of further abscess formation. She was sent to Leysin under Dr. Rollier. In February, 1927, she lost all power and sensation in her legs. After about a fortnight of complete paraplegia there is said to have been a gradual improvement, but in January, 1930, Rollier reported that the paraplegia had regressed.

Sept. 6, 1930.—Admitted to Wingfield Orthopædic Hospital. General mid-dorsal kyphosis. Complete motor, sensory, and sphincter loss below D.7. Spasms present.

Sept. 12.—*Laminectomy, bone-graft, costotransversectomy*—finding the theca was compressed by an encapsuled protrusion of caseous débris which had pushed its way backwards in the left side of the theca.

Comment.—The following points are noteworthy :—

1. Early girdle pains, ten months before a diagnosis of spinal caries.

2. After two years and nine months on her back the patient was allowed up, but within three months it was evident that the disease was still active. She was put on her back again, but in February, 1927—four years and three months after her first symptoms—complete paraplegia supervened.

3. This paralysis had persisted with temporary improvement for a further three years and seven months before any attempt at decompression.

4. It is a paraplegia of late onset, but an operable cause of pressure was found.

5. An operation so late in the course of paraplegia has relatively little chance of success.

TECHNIQUE OF THE COMBINED LAMINECTOMY AND GRAFT OPERATION.

The patient is anæsthetized on his frame, or in cases of sharp angular kyphos on his plaster bed ; then his turning-case is put on, strapped into place, and he is turned (*Figs.* 131–133). The anæsthetist arranges the flannel bandages between the anæsthetic bars so as to support the patient's forehead. It is very important for the anæsthetist at this stage to make sure that there is no edge of the turning-case pressing on the patient's neck or axilla. In this operation, amongst others in which a good deal of the body is covered only with sterile towels, we use double towels containing a layer of gamgee tissue between them in order to keep the patient warm.

The incision is slightly to the left side so as to keep the scar away from the spinous processes, and in order that the motor-saw may be used unhampered by retractors on the surgeon's side. The skin and subcutaneous tissue

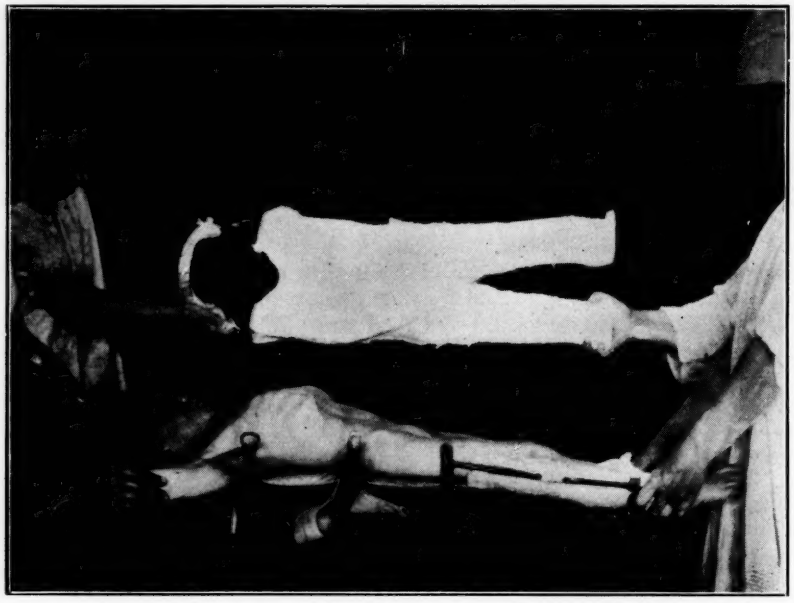

FIG. 131.—Child on frame. Turning-case being applied.

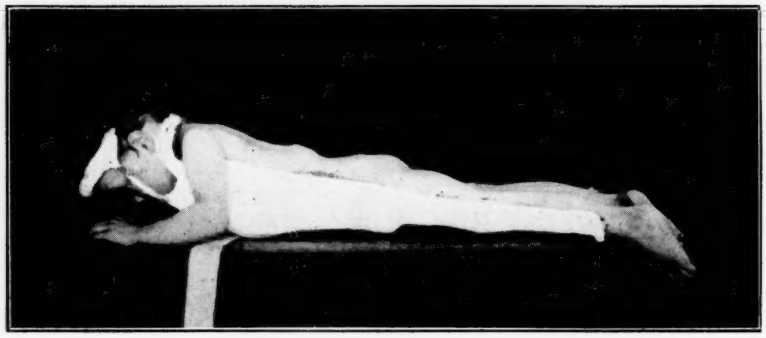

FIG. 132.—Child lying on turning-case as for operation.

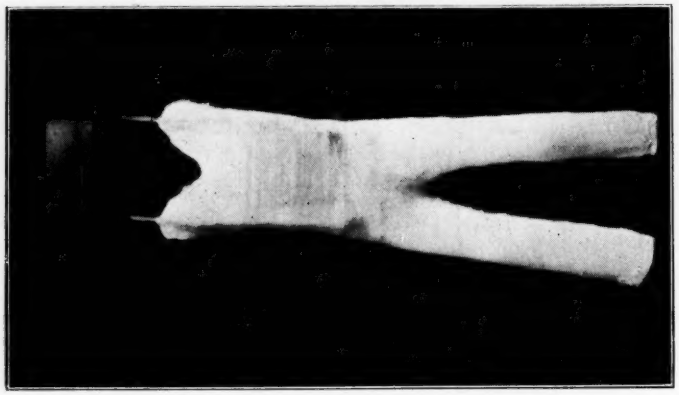

FIG. 133.—Turning-case fitted with anæsthetic extension.

are lifted from the deep fascia and reflected away from the surgeon beyond the line of spinous processes; the skin edges are closely and smoothly covered with fine towels.

Then an incision is made on to the apex of each spinous process and carried by a dip of the point of the knife through the interspinous ligaments over an area covering the three spines of the laminectomy area, two spines above, and two below. As the line of processes is often deranged by caries, and as it is best to make the incision run exactly over the apex of each process, it is helpful to define each process as the incision reaches it by gripping it between the points of a pair of dissecting forceps held in the left hand. The motor-saw is then taken and two incisions are made into each spinous process forwards and slightly outwards, to right and to left. These saw cuts are clearly shown on *Fig.* 134. They start in the knife cut and separate a thick flake of bone from the spinous process on each side. When each process has been dealt with in this way the saw is laid aside and an osteotome, 1½ in. wide, is used to complete the separation of the lateral flakes of bone from the central part of each spinous process, and then to carry these flakes outwards, together with the periosteum of the bases of the spines and laminæ. I use an osteotome very broad so that it cannot possibly get between the laminæ, and sharp so that it can be used gently without disturbing the carious centra. Each time the osteotome travels outwards the space between it and the spinous process is packed with gauze, so that by the time the upper end is reached there is a thick packing all the way up that side. The other side is then treated in the same way (*Fig.* 135). It should be said that the laminæ are only fully exposed where they are to be removed. In the case of the two spines above and below, the displacement of the flake of bone and periosteum does not go beyond the base of the spinous process.

FIG. 134.—Showing motor-saw cuts into a spinous process.

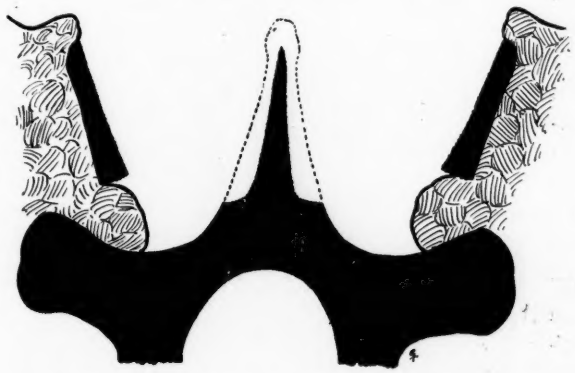

FIG. 135.—State of affairs after reflection of osteo-periosteal flaps in the area of the laminectomy.

The laminectomy is then done. It is essential to make the laminectomy opening of full width and long enough to relieve all the pressure present or at all likely to occur. *Case* 1 is an instance of a laminectomy opening that was not long enough, though now I realize that the recurrent paraplegia could and should have been avoided by a costotransversectomy done before, or a week after, the laminectomy. I do not think I have ever had to remove

more than three laminæ for Pott's paraplegia. I do not open the dura, but 'seek' gently round it and sometimes evacuate a mass of débris from the side or the front of the theca.

Next comes the graft. The length and shape of the grafts required are recorded by bending a probe, and two 'Albee' grafts about $\frac{3}{8}$ in. wide are cut from the tibia. These can generally be straight. If so, they are put in with the periosteal surface deep—that is, with the smooth limiting membrane turned towards the theca. If the spine is straight or only moderately kyphotic, this gives plenty of clearance at the site of the lesion, because the grafts are carried fairly high up on the spinous processes above and below. If, on the other hand, there is much angular curvature, one of two methods must be used :—

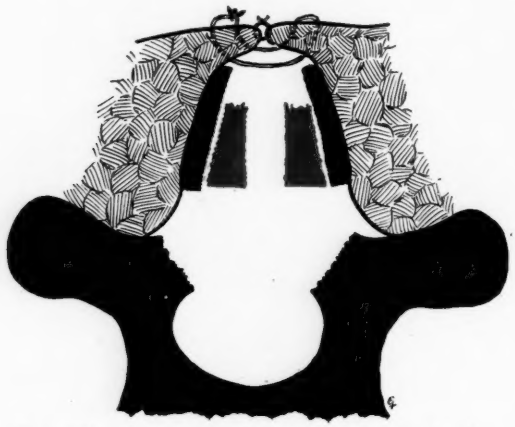

FIG. 136.—Grafts bridging defect left by laminectomy, with their lateral osteogenetic hosts.

1. *Shaped grafts*, to fit the angulation. This means using the broadest part of the tibia, and not sparing the crest.

2. *Flexible grafts* (when a graft of sufficient length and angulation is unobtainable), prepared either by the earlier method of multiple transverse cuts about $\frac{1}{10}$ in. apart on the medullary side of an Albee graft, and for this a very fast running saw is advisable; or by a technique in which one makes a number of parallel longitudinal cuts into the tibia $\frac{1}{10}$ in. or less apart; after a transverse cut at each end six or more very thin and fairly flexible Albee grafts can be lifted from the tibia. Three or four of these are used on each side like laminated springs flexed to fit the angulation and firmly fixed to the spinous processes above and below. (I use a number of separate leaves. Albee, when lecturing at the Orthopædic Section of the Royal Society of Medicine in April, 1930, showed a lantern slide illustrating a graft in which a number of longitudinal incisions had been made but in which both ends were solid. In this case the range of flexion of the graft must be very small.)

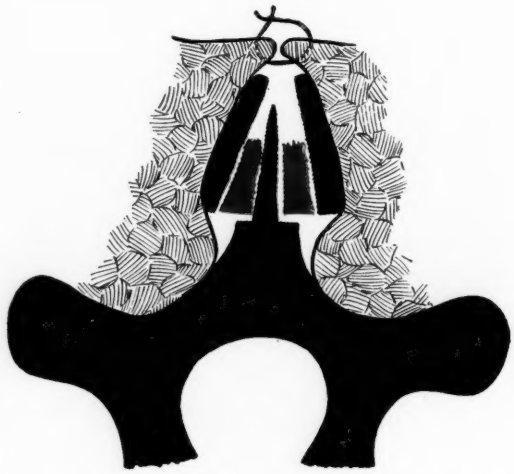

FIG. 137.—Showing grafts as supported on spines above and below area of laminectomy.

When the grafts are firmly in place, and one has made sure that there

is plenty of space between their deep surface and the theca, the edges of the supraspinous ligament, carrying with them the lateral flakes of bone from each spinous process, are sewn together over the grafts (*Figs.* 136–138). Just previous to this, if there are one or more prominent spinous processes, they are nipped across with bone-cutting forceps and bent under the suture line. This makes for comfort and safeguards the skin from pressure. The operation is completed by suture of the skin of the back and the leg. Then dressings and carefully graded layers of wool are applied, the frame or plaster bed is laid in position, the straps are pulled up, and the patient is re-turned.

After-treatment.—The patient remains on his frame or plaster bed for three or four months. He is turned whenever necessary by the method described (the routine in *all* cases of spinal caries), and each time the utmost care must be devoted to the exact adjustment and soft smooth padding of spinal frame or plaster bed.

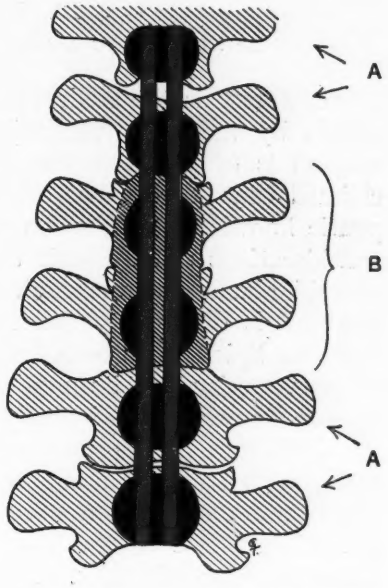

FIG. 138.—Diagrammatic coronal section of spinous processes and graft, with vertebræ and area of laminectomy indicated. A, Grafts well supported on spines; B, Area of laminectomy.

Finally I would emphasize three things :—

1. I believe that if laminectomy is to be employed for Pott's paraplegia it should always be supplemented by grafting.

2. Good technique of such grafting is vital, for the grafts have to bridge the laminectomy opening without absorption, and because the strain on grafts in Pott's disease is often great. Therefore the grafts must be strong and be given a vascular and osseous bed or there will be risk of their absorption or fracture. It is for this purpose that I separate from each spinous process lateral flakes of bone with their periosteum intact, and a good vascular supply from the erector spinæ. At the end of the operation these lie with their raw surfaces in contact with the grafts; thus they provide an almost continuous series of vascular ossific nodes ready to vitalize and recolonize the grafts.

3. The continuous immobilization of the whole spine before, during, and after operation.

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SIALOGRAPHY: ITS TECHNIQUE AND APPLICATIONS.

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Sialography is the radiographic demonstration of the ducts of the salivary glands by means of the injection of substances opaque to X rays. The method is of necessity one of strictly limited applicability, yet it may be claimed that its use has added to our pathological, diagnostic, and therapeutic knowledge of diseases of the salivary glands. In practice, anatomical considerations usually limit the investigation to a study of the ducts of the parotid gland, and although it has also been applied to the submaxillary gland, it is difficult to believe it can often be of use here. As an opaque medium, lipiodol has been found to be the most satisfactory, for owing both to its viscosity and its non-irritating nature it is less stimulating than the opaque bromides and iodides, which tend to be too rapidly ejected.

Although in the past sialography had often been carried out in the cadaver, Bársony,[1] writing in 1925, was the first to publish a case report where the method had been employed. Using a solution of 20 per cent potassium iodide he was able to demonstrate an enormous degree of dilatation of one parotid duct. This solution, however, gave rise to a considerable amount of pain. Of later workers, both Carlsten[2] (1926) and Keith[3] (1928), using lipiodol, have each recorded a case. The only record in the literature of the method as applied to the submaxillary duct is a scanty communication of Wiskovsky's[4] (1927).

Indications.—Briefly, sialography will demonstrate abnormalities, dilatations, and obstructions of the larger and the smaller parotid ducts. It may, therefore, give evidence of a positive or negative value. For example, in the case of parotid calculi situated in the intraglandular part of the duct, radiography is often uncertain, and the employment of sialography may aid in exact localization. In the group of cases which fall under the heading of recurrent subacute parotitis of non-calculous origin—a condition which is usually a recurrent sialodochitis—the method may demonstrate certain well-defined lesions of the smaller ducts. Fistula cases, though rare in civil surgery, often present problems of a diagnostic and surgical nature which can at times be solved by sialography. Finally, the method may be employed for its negative value in such cases as true Mikulicz disease.

Further, it may be pointed out that whilst catheterization of the parotid ducts has an established diagnostic value, it is possible that the technique of sialography may possess a therapeutic value in the introduction of medicated substances into the salivary ducts direct.

Technique.—Sialography is not a difficult process, but is one requiring care and gentleness. From the point of view of the patient it is usually

painless, and the use of a local anæsthetic has the objection of tending to dry up all salivary secretions, thus rendering the entry of the duct more difficult. Only in one case (*Case* 2) was the completion of the injection followed by a transient swelling of the gland with some slight discomfort. At the end of twenty-four hours or less all the lipiodol has disappeared from the parotid ducts.

Apparatus.—In carrying out the actual injection, home-made apparatus consisting of a rubber fountain-pen bulb attached to a short length of glass tubing, one end of which is drawn out to a fine point about 1 mm. in diameter, is the most satisfactory. It is usually best to have a series of these of graduated sizes. The glass tubing may either be straight or bent at a slight angle as shown in *Fig.* 139. The apparatus is sterilized either by boiling or by immersion in spirit before use. Round-end needles of No. 17 gauge and short lengths of fine ureteric catheters have both been tried, but the former is rather liable to damage the delicate structures of the parotid duct, and the latter is somewhat difficult to introduce. Further, it has been found much easier to steady in the duct the very light apparatus described, than to employ either of the other methods, which necessitate the attachment of a comparatively heavy Record syringe.

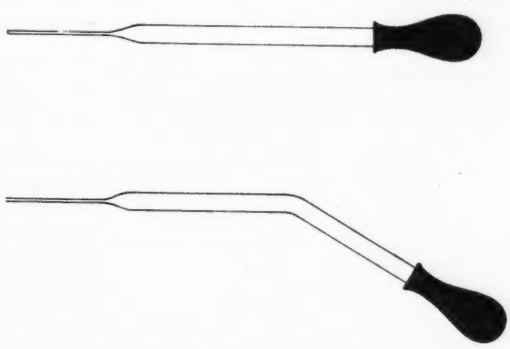

FIG. 139.—Type of syringe used for sialography.

The Patient.—Before beginning the process the patient washes out the mouth with water or a mild antiseptic. The orifice of the duct is then sought in the region of the second upper molar tooth, and gentle massage of the parotid will usually render it patent. If necessary, one or two of the graduated cannulas may be passed into it to dilate it slightly. In cases where the duct orifice cannot be identified, or where it is impossible to pass even the finest cannula, the patient is given a piece of lemon to suck. After this has been done there should be no difficulty in localizing the orifice.

The Injection.—The patient thus prepared lies on his back on the X-ray table with his head turned towards the sound side. The apparatus is filled with lipiodol, and the cannula introduced into the duct for about half an inch. Once in position, the apparatus should be held extremely lightly in the hand. The bulb is then squeezed gently for about half a minute to force the lipiodol into the ducts. Screening is then carried out, and if the injection has been satisfactory, the main duct and the larger branches can be identified. If this is the case, the sialogram is taken. The injection process is then repeated, to be followed by a further screening and picture. As a rule $\frac{1}{2}$ to 1 c.c. of lipiodol is all that is required. The development of pain over the parotid region or swelling of the gland indicates that no more lipiodol should be injected. Should these symptoms develop, they can be quickly relieved by massage carried out to empty the gland or duct.

CASE REPORTS.

Case 1.—**Glandular fistula (right parotid).**

HISTORY.—E. M. C., a housemaid, age 45, attended St. Bartholomew's Hospital on March 6, 1930, complaining of a profuse discharge of watery fluid from below the right ear during meals. In 1927 she had been operated on for two abscesses in the right parotid region, one being just in front of the ear, and the other over the lower part of the mastoid process. The incisions healed up in the course of a few days. From the history it was impossible to be certain whether these lesions were due to suppurative lymphadenitis, or whether they were the result of suppurative parotitis. In January, 1929, she started to get a discharge from the lower part of the mastoid scar, which had gradually increased in amount. The discharge was always of a watery nature, and only occurred during meals, but at these times it was so profuse as to necessitate the patient's wearing a towel round the neck. Mumps had occurred during childhood.

PHYSICAL SIGNS.—The posterior scar was situated over the lower part of the mastoid process. At its lower end there was a pin-point opening from which saliva poured profusely when the patient was given a lemon to suck. The right parotid duct was normal and secreted freely, and no abnormality could be made out in the parotid gland itself.

FIG. 140.—*Case* 1. Sialogram. The opacity over the upper and posterior part of the second cervical spine corresponds to the opening of the fistula.

INVESTIGATIONS.—Ordinary skiagrams of the right parotid region failed to show any evidence of calculi. Sialography was carried out on March 20 to determine whether or not there was any communication between the fistulous track and any parotid duct of appreciable size. The sialogram (*Fig.* 140) shows the main duct of the gland, the larger branches, and the ductules, all well filled with lipiodol. The site of the fistula is marked by the opacity at the upper and posterior part of the second cervical spine, and there is no communication between this and the parotid ducts.

TREATMENT.—On May 12 the fistula was treated without anæsthesia by means of the electrocautery. The discharge of saliva ceased in about a week, and there has been no recurrence since then.

Comment.—In the usual classification of parotid fistulæ into gland and duct types, the distinction is one based entirely on topographical anatomy. Further, this distinction is one which profoundly affects the surgical principles applicable to two types of fistula. The more exact knowledge, therefore, gained by sialography in showing the relationship between a fistula and the parotid ducts has a direct bearing on the question of treatment, especially where a so-called glandular type of fistula communicates with a comparatively large duct.

Case 2.—**Recurrent subacute parotitis (right.)**

HISTORY.—R. P., female, age 23, attended St. Bartholomew's Hospital on Oct. 16, 1930, complaining of recurrent attacks of swelling in the right parotid

region. The condition first started in 1927, the swelling always appearing during a meal, and once it was present lasting from one to seven days. The longest period of freedom from an attack had been six months, but during the past twelve months the attacks had been getting much more frequent. When the swelling was present it was painful, but not severely so, though at times it had been bad enough to make the patient stop eating, and had prevented opening of the mouth more than half way. The last attack started on Oct. 6, and the swelling increased a little with each meal, to diminish somewhat between meals. It finally cleared up on Oct. 10. There had at no time been any discharge into the mouth, though at times during the past year there had been a bad taste. At the age of 13 the patient had mumps.

PHYSICAL SIGNS.—No visible swelling of the right parotid region was present. On palpation there was a rather ill-defined swelling about an inch in diameter with its upper limit three-quarters of an inch below the zygoma and at the level of the external meatus. The swelling was situated over the masseter, and its forward limit corresponded to the anterior limit of the muscle. Clinically most of the swelling appeared to be rather above the level of the normal parotid duct. The orifice of the duct was normal, as were the orifices of all the other salivary glands.

FIG. 141.—*Case* 2. Sialogram showing the minute spherical dilatations towards the terminations of the smaller ducts in the anterior part of the gland.

INVESTIGATIONS.—Catheterization of the right parotid duct was carried out after the patient had been given a lemon to suck. The saliva which was obtained contained several elongated shreds of highly tenacious mucus. Examination of these shreds and also of the more fluid part of the saliva, by means of a Gram film, showed that pus cells were present from one to three per field. (The presence of pus cells in a catheter specimen of saliva is always definite evidence of an inflammatory lesion of the gland or ducts.) Skiagrams of the right parotid region showed no calculi. On Oct. 17 sialography was carried out without difficulty. Towards the end of the examination the patient complained of slight discomfort over the parotid region and there was at the same time a little fullness to be made out. The latter had entirely cleared up when the patient was seen again at the end of twenty-four hours. The sialogram, reproduced in *Fig.* 141, shows good filling of the parotid duct and its branches. In the anterior part of the gland there are in addition minute spherical dilatations present towards the terminations of the ductules—suggesting a 'bronchiectactic' condition. A further interesting feature is that the area where these dilatations are present corresponds to the position of the palpable swelling of the parotid.

Comment.—Recurrent subacute parotitis when not associated with the presence of calculi is invariably due to a mild degree of infection of the parotid ducts. The secretion in such cases is excessively mucoid in type, and at times plugs of mucus and débris corresponding to the main duct or its larger branches may be obtained by massage of the gland or by catheterization. Normally, parotid secretion is almost free from mucus, but traces are derived from glands situated in the larger ducts. In inflammatory

conditions this mucoid secretion is markedly increased, and together with pus cells and epithelial cells forms plugs which actually obstruct the ducts, and thus dam back the normal glandular secretion. In the present case the recurrent duct obstruction was undoubtedly the factor bringing about the dilatation of the smaller ducts in the anterior part of the gland.

Treatment in such cases should be directed towards the clearing up of all possible sources of buccal infection, and in addition, as a prophylactic measure, the patient should be shown how to massage the parotid gland and its ducts, in order to clear them of tenacious secretion. This should then be carried out for about five minutes before and after every meal.

Case 3.—**Parotid calculus (left).**

HISTORY.—E. M., age 34, male, clicker, attended St. Bartholomew's Hospital on April 14, 1930, complaining of a swelling of the left parotid region and a discharge into the mouth. In 1927 the patient first began to get a swelling of this region which came up during meals and then slowly went away afterwards. On the first occasion these symptoms were present for about a fortnight, and ever since then at intervals of a few weeks there had been attacks of a similar nature. In August, 1929, the swelling of the left parotid became very large and painful, and at the same time an ulcer developed inside the mouth in the neighbourhood of the duct, from which large amounts of foul pus poured. After a week the parotid swelling receded. Two weeks later the patient was operated on at Northampton General Hospital, for a small residual swelling near the angle of the jaw. No calculus was found and the wound healed by first intention. Ever since that time there had been swelling of the gland with every meal and a continual discharge of pus into the mouth.

PHYSICAL SIGNS.—About one inch above and in front of the angle of the jaw on the left side there was a slight visible swelling. Palpation revealed a small swelling about $\frac{3}{4}$ in. × $\frac{1}{2}$ in., firm in consistency, slightly tender, and not fixed to adjacent tissues. The duct orifice was reddened and slightly ulcerated, and pressure over the parotid gave rise to a flow of mucopus.

FIG. 142.—*Case* 3. Skiagram showing a calculus in the posterior part of the left parotid region. Its position is marked with the white dot.

FIG. 143.—*Case* 3. Sialogram showing dilatation of the main parotid duct and absent filling of the branches. The position of the calculus corresponds to the ovoid dilatation at the posterior limit of the dilated duct. A certain amount of lipiodol is present in the floor of the mouth.

INVESTIGATIONS.—Examination of a catheter specimen of the pus showed the presence of pus cells in very large numbers, and on culture a pure growth of

Staphylococcus aureus was obtained. Ordinary skiagrams showed a small calculus in the posterior part of the gland (*Fig.* 142). On April 14 sialography was carried out without difficulty, and in *Fig.* 143 the marked degree of dilatation of the main parotid duct is shown. At the extreme posterior limit of the duct there is a dense ovoid shadow which corresponds to the position of the calculus, and practically no lipiodol has got beyond this point. The other interesting feature is that none of the smaller ducts shows any filling at all. There is a certain amount of lipiodol present in the floor of the mouth.

TREATMENT.—On April 16 a small almost spherical calculus 5 mm. in diameter was excised from a markedly dilated duct. The wound healed by first intention, the discharge of pus into the mouth ceased in about a week, and the patient has remained free from symptoms since then.

Comment.—The points of interest in the investigation of this case are: (1) The degree of dilatation of the main parotid duct; and (2) The absent filling of all the branches of the duct. The explanation of the latter is probably that the preceding obstructive lesions followed by an acute inflammation had led to their obliteration.

Case 4.—**Mikulicz's disease.**

HISTORY.—A. E., male, age 48, attended St. Bartholomew's Hospital on March 12, 1929, complaining of swellings on both sides of the face and neck. About Christmas, 1928, the patient first noticed swellings in the parotid and submaxillary regions on both sides. Since they appeared they have tended gradually to increase in size, though from time to time the patient stated that they had varied somewhat. There had been no discomfort, and no disturbance of the general health.

PHYSICAL SIGNS.—Both parotid glands showed a visible enlargement of moderate degree, but did not overstep the normal anatomical limits. The overlying skin was healthy, the consistency firm, and no tenderness was present. The submaxillary glands were both enlarged to three times the normal size, but were otherwise normal. Neither the sublingual nor the lacrimal glands showed any enlargement.

INVESTIGATIONS.—Sialography of the right parotid gland was carried out on April 12 and showed no abnormality (*Fig.* 144). Ordinary skiagrams of the affected regions showed no abnormality. In addition, complete examination of the blood, including the Wassermann reaction, proved negative.

FIG. 144.—*Case* 4. Sialogram of right parotid in a patient with Mikulicz's disease proper.

Comment.—In this case all the diseases known to give rise to Mikulicz's syndrome were excluded, and the condition was regarded as a true example of Mikulicz's disease proper of the non-familial type. The sialogram proved in addition that this disease is not associated with any gross lesion of the parotid ducts.

SUMMARY.

1. The technique of sialography of the parotid, using lipiodol as a medium, is described, and it is shown to be a simple and a safe procedure.

2. By means of its application the normality or otherwise of the parotid duct and its branches can be demonstrated, and in pathological conditions affecting the gland the information so gained may render diagnosis more exact, and consequent treatment more rational.

3. Four cases are described where sialography has yielded useful information. In the first it was possible to demonstrate that a parotid fistula was strictly glandular in type; in the second a pathological dilatation of the smaller ducts was shown following recurrent subacute parotitis; in the third the markedly dilated condition of the whole of the parotid duct was shown in association with a calculus, together with absent filling of the smaller ducts; and in the fourth case the normality of the ducts was demonstrated in a case of Mikulicz's disease.

My thanks are due to Sir Holburt Waring, to Mr. L. Bathe Rawling, and to Mr. W. Girling Ball for allowing me to record these cases.

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TWO CASES OF CARCINOMA OF THE SMALL INTESTINE.

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THE following cases are reported on account of the interesting contrast which they afford.

Case 1.—Male, age 54. The illness commenced with vomiting after the taking of rich food three months previously, and in the intervening period the patient had vomited every second day. When seen his appetite was poor, he had marked flatulence, and a feeling of fullness. His bowels had been kept regular by medicine. He had lost 3 or 4 lb. in weight.

The only positive finding on examination was slight fullness in the epigastric and umbilical regions. Gastric analysis showed nothing abnormal. X-ray examination revealed delay in emptying of the stomach, and marked dilatation of

FIG. 145.—*Case* 1. Carcinoma of small intestine.

the small bowel with stasis. X-ray examination, then, pointed to obstruction in the ileum.

At operation the stomach and upper part of the small intestine were much distended; half-way down the small intestine there was a hard ring stricture causing almost complete stenosis (*Fig.* 145). In the mesentery adjoining the stricture there were two slightly enlarged glands. There was no evidence of metastases in other lymphatic glands, in the peritoneum, or in the liver. Resection was carried out, and the patient made a normal and uneventful recovery. Six months later he had gained 14 lb. in weight.

Examination of the bowel wall at the site of the stricture revealed an adenocarcinoma with considerable hyperplasia of fibrous tissue (*Fig.* 146). The lymph glands showed no metastatic deposits.

FIG. 146.—*Case* 1. Adenocarcinoma of small intestine, showing a more or less normal mucous membrane overlying the cancerous growth.

Case 2.—Male, age 57. The patient came complaining of a swelling above the right clavicle which he had noticed for the first time a month previously. He had been treated for neuritis, but this pain had largely disappeared.

Physical examination revealed a mass of enlarged glands in the supraclavicular region, and another mass in the right axilla. Hospital treatment was refused. A week later the man presented himself again, complaining of increasing pain and stiffness, and the glands were larger. Three days later he was admitted to hospital with several curious additions to his symptoms, a gradually increasing paralysis of the right side together with speech difficulty. Physical examination revealed a right-sided hemiplegia, flaccid in type, retention of urine, incontinence of fæces, and a complete motor aphasia. The glands in the supraclavicular region, however, were distinctly smaller. The patient became comatose and died.

At autopsy the jejunal wall, four feet below the duodenojejunal junction, showed externally a round flattened pigmented plaque, about an inch and a half in diameter. On opening the gut an ulcer with necrotic interior and rounded edges presented itself, but the lumen of the gut was unaltered in diameter (*Fig.* 147). The mesenteric glands near to the ulcer were enlarged and partly necrotic. The liver was large and riddled with growth, which was partly pigmented. The spleen was also entirely infiltrated with metastatic growth, the nodules being of varying size, and partly pigmented. Both lungs had scattered metastatic deposits, and the kidneys also showed a few nodules. The glands in the right supraclavicular triangle showed growth, and in the occipital lobe of the left cerebral hemisphere there was a large area of new growth, both necrotic and hæmorrhagic.

Sections of the edge of the primary ulcer (*Figs.* 148, 149) showed a more or less normal intact mucous membrane overlying a nodule of cells whose character was quite unlike that of glandular cells. These cells were oval or polyhedral, unequal in size, and of fairly uniform appearance. In places there was a suggestion of acini, but a suggestion only. The metastases showed a similar structure.

COMMENT.

The two cases reported afford an interesting contrast in history, symptoms, microscopic histology, and malignancy. In the first case symptoms had shown themselves over a period of months. Vomiting had been more or less constantly present, appetite had been impaired, and the patient had lost

FIG. 147.—*Case* 2. Carcinoma of small intestine.

weight. The second patient had not suffered any ill health, and presented himself only shortly before his death because of the lumps in his neck. He did not call attention to his abdomen at all.

The first patient's cancer went on to stricture of the small gut with almost complete blocking. The second case, apart from the small localized ulcer, presented no alteration in the lumen of the gut.

The pathological histology of the first case was a typical very scirrhous adenocarcinoma. The histology of the second case suggests that the primary

growth may have arisen in a carcinoid of the jejunal mucous membrane at this point. As stated above, no glandular structure was observed, and there was a minimum of intercellular tissue.

The malignancy of the two cases also stands out in marked contrast.

FIG. 148.—*Case* 2. Carcinomatous change in carcinoid (?) More or less normal mucous membrane overlies the cancerous growth.

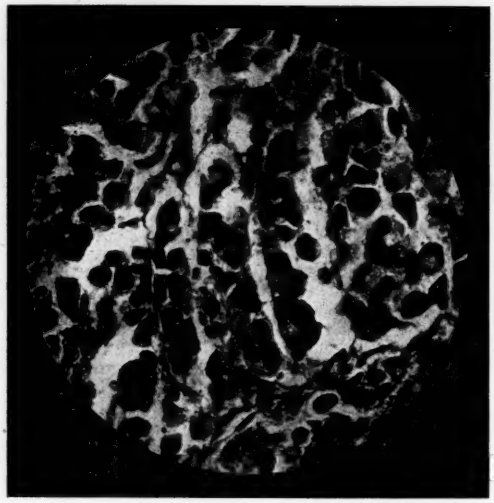

FIG. 149.—*Case* 2. High-power view of the cancer shown in *Fig.* 148.

In the first no metastases were discovered. The glands neighbouring the growth were not involved. In the second case, though the primary growth was so comparatively small, the metastases were widespread, glands, liver, spleen, lungs, kidney, and brain all being involved.

SHORT NOTES OF RARE OR OBSCURE CASES

A TUMOUR OF THE CAROTID BODY.

By LAURENCE O'SHAUGHNESSY, Omdurman.

The patient, a well-developed Sudanese of about 25 years, complained of a swelling in his neck (*Fig.* 150). He had been conscious of this for many years, but a rapid increase in size during the last few months had caused him to seek advice. Diagnosis seemed to lie between carotid tumour and parotid tumour, but the absence of pain and the position of the swelling favoured the former.

Operation.—The common carotid artery was ligatured with catgut, and by extending the incision the tumour was exposed. It was on the whole easily enucleated; only at its upper pole, which extended to the base of the skull, was any difficulty experienced. Separation from the carotid sheath was easily effected, although this was a point at which much difficulty had been anticipated. The wound was closed, and the patient made a good post-operative recovery. On the following day he was found to have a facial paralysis on the side of the operation.

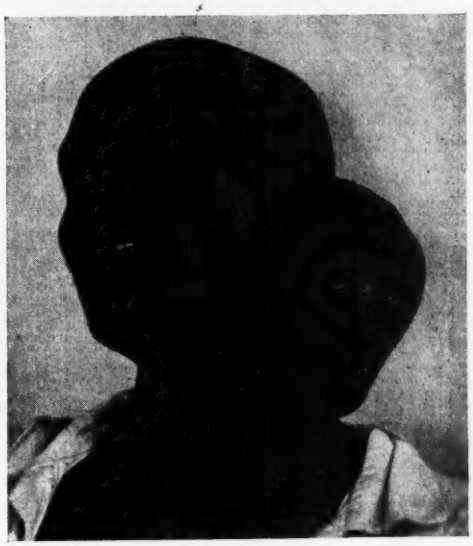

Fig. 150.—Tumour of the carotid body.

Pathological Report.—I am indebted to Dr. E. S. Horgan for the following report:—

"The tumour was roughly oval in shape (15 cm. by 11 cm.), solid in consistency, with a well-marked fibrous capsule. On section it showed a series of irregular lobulated masses, gelatinous in appearance and of a greyish-white colour. In the centre of one was a small hæmorrhagic area.

"*Microscopical Examination.*—Low power (*Fig.* 151): The general structure consisted of solid masses of cells, which in some cases showed an alveolar or whorled structure; but such 'whorls' were ill-defined, and in many places the cells had no definite arrangement. The tumour had undergone extensive mucoid degeneration, and large areas consisted of myxomatous tissue with islands of tumour cells.

"High power (*Fig.* 152): The cells varied in shape and size, ranging from large polyhedral or spheroidal cells with well-marked granular cytoplasm

to smaller, more darkly staining spindle cells, some of which appeared definitely sarcomatous. There were numerous dilated capillaries present, but they appeared to bear no definite relation to the cell masses. Nor was it possible to trace any relation between their lining endothelial cells and the large spheroidal tumour cells.

FIG. 151.—Low power. (× 65.)

"No pigment could be demonstrated in any part of the tumour.

"The long history of growth, the close relations with the carotid sheath, the well-marked encapsulation, and the general histological picture would suggest that this was a tumour arising from the carotid body.

"In the past various names like 'perithelioma' have been given to such tumours, but these serve merely to confuse the issue. While the genesis and evolution of this neoplasm remain unknown it seems better to avoid labels."

FIG. 152.—High power. (× 230.)

Comment.—The mortality of this operation is high; Licini[1] gives it as 35 per cent. Beavan and McCarthy[2] consider that operation is not justifiable, and in their case employed radium with success. They state that only a small proportion of these tumours is malignant, and attribute the operative mortality largely to ligature of the carotid. My own experience of carotid ligature did not lead me to fear its effect, and, in the absence of radium, the only treatment for a rapidly growing tumour was extirpation.

I am indebted to the Director of the Sudan Medical Service for his permission to publish this note.

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[1] LICINI, *Zentralb. f. Chir.*, 1908, xcvi, 327.
[2] BEAVAN and MCCARTHY, *Surg. Gynecol. and Obst.*, 1929, xlix, 704.

ANTERIOR DISLOCATION OF THE RADIUS AND ITS RECURRENCE.

By C. H. CORBETT,

ASSISTANT SURGEON, THE ROYAL INFIRMARY, HULL.

The mechanism producing anterior dislocation of the radial head seems to be almost as great a riddle as that of anterior dislocation of both the radius and ulna at the elbow. Cohn[1] in 1922 found that not much could be added to the findings of Colson and Huguie's work of nearly a century ago, which were that anterior dislocation of both bones at the elbow was due to one of three causes: (1) Forced flexion of the forearm; (2) Upper arm fixation and turning the forearm round the forearm axis; and (3) Hyperextension of the forearm. In the mechanism of anterior dislocation of the radial head alone it seems probable that one or more of the above factors should exist. Cochrane[2] is of the opinion that the second and third factors are involved in anterior dislocation of the radial head, which is, he considers, due to a rotation of the forearm (radius) into pronation (i.e., 'rotation round the axis of the forearm' —No. 2 of Colson and Huguie's causes) plus hyperextension of the radial head through a fulcrum produced by an impinging of the radius upon the ulna immediately distal to the level of the radial tubercle. In the case set out below, the factor producing hyperextension was applied upon the distal end of the radius by the fall of the body thereupon, the arm being in pronation at the time. Added to and aiding these two factors, however, was the position of the forearm at right-angle flexion. Thus we see in the case in question the three factors involved were: (1) Flexion of the forearm; (2) Upper arm fixation and pronation of the forearm; and (3) Hyperextension of the radial head through a fulcrum produced by an impinging of the radius upon the ulna, probably immediately distal to the radial tubercle, and the force being applied by the weight of the body falling on the distal end of the radius. Thus we find that the general conclusions of Colson and Huguie nearly a century ago still hold.

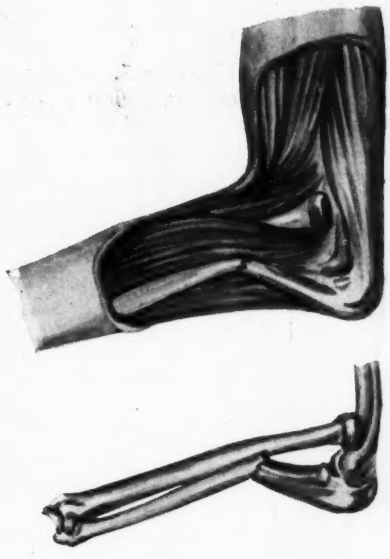

Fig. 153.—Fracture of the ulna in the upper third with dislocation of the capitellum (after Helferich), due to a force applied at the site of fracture and continued to produce dislocation.

Thomas's[3] view is very different, for in his discussion upon elbow fractures and dislocations, he says that the mechanism of anterior dislocation of the radial head is as follows: "If the elbow struck on an underlying stone or other such object, and this were of such size and shape and were so situated

that only the radial head received the impact upon it, we can easily imagine the force driving the radial head into anterior dislocation." It is difficult to imagine the conditions occurring which Thomas lays down for this dislocation —indeed, in the case under review the boy fell upon the smooth wooden floor, and though this type of dislocation is infrequent, it is common enough to suggest that other factors as suggested by Cochrane are usually at work. When one studies, however, the case of dislocation of the radius with fracture of the ulna, one sees a mechanism (*Fig.* 153) similar to what Thomas imagined might occur—namely, a fall on to the back of the flexed forearm which fractured the ulna at the junction of the middle and upper third and dislocated the radial head forwards. It must necessarily be an infrequent mode of dislocation, as the olecranon is such an efficient barrier and guard.

Scudder[4] does not publish his views upon the mechanism of this dislocation.

CASE REPORT.

H. B., male, age 5. The patient was playing with some other children when he slipped upon a wooden floor and fell with his left arm bent under him, with a resultant dislocation of the radial head, his elbow being in flexion, the forearm in pronation, and the body falling over the distal end of the forearm. The dislocation was reduced by Dr. Kirk of Barton-on-Humber and kept in a sling for fourteen days.

The accident occurred on Oct. 19, 1927, and thereafter with trivial injuries there were subsequent redislocations on Jan. 24, 1928, Oct. 17, 1928, Nov. 15, 1929, and in January, 1930.

Broadly speaking, injury to the elbow region due to a comparatively slight accident such as a fall occurs in young subjects; even such an unusual accident as anterior dislocation of the forearm bones at the elbow occurs for the most part in subjects under 16 years of age, and in older people it is usually a very severe type of accident (a fall from a height, entanglement in a machinery belt, etc.) which causes the condition.

The boy in question was seen by me at the Surgical Outpatients' Department, Royal Infirmary, Hull, and was admitted to the wards on Feb. 13, 1930. It was judged that the orbicular ligament was beyond ordinary repair and that a plastic operation of some kind was indicated.

Campbell,[5] in 1929, discussing unreduced dislocations at the elbow, mentions the lack of particular satisfaction in obtaining fascia near the site of the dislocated radius, and ingeniously takes a strip of fascia lata, drills a hole in the ulna at the level of the radial neck, passes the fascial strip through the hole and around the neck, and sutures the orbicular ligament. It is to be doubted whether this is a facile procedure in the young subject and whether it is altogether necessary.

FIG. 154.—The line of incision.

OPERATION (*Figs.* 154–157).—In the case under review it was considered that, given fascia stout enough for orbicular ligament repair and for strengthening of the stretched anterior capsule, the radial head should maintain its position, and it was hoped that the procedure of transference of part of the lacertus fibrosa (bicipital fascia) would meet the case (*Fig.* 155).

An incision (*Fig.* 154) was made parallel and external to the bicipital tendon and along the outer line of its fascia. The lateral cutaneous nerve was exposed and retracted, the cubital vein partially resected and ligated, the deep fascia

divided, and the bicipital tendon with the lacertus fibrosa defined. The brachio-radialis and extensor carpi radialis longior were brought into view and retracted, and the radial nerve thus exposed was retracted laterally with the above muscles. The supinator brevis was exposed and retracted downwards and outwards, and the capsule of the joint and the orbicular ligament were brought into the field. The external lateral ligament was defined in the lower three-quarters of its extent. The orbicular ligament was found torn and was fairly widely separated (*Fig.* 156).

At this stage a suitable strip of the lacertus fibrosa of about $\frac{3}{4} \times \frac{1}{2}$ in. was taken and stitched with interrupted silk to the lateral ligament, capsule, and orbicular ligament as in *Fig.* 157.

Subsequent History.—The patient's elbow was maintained extended for a week, when light passive movement and massage were inaugurated, and rotation

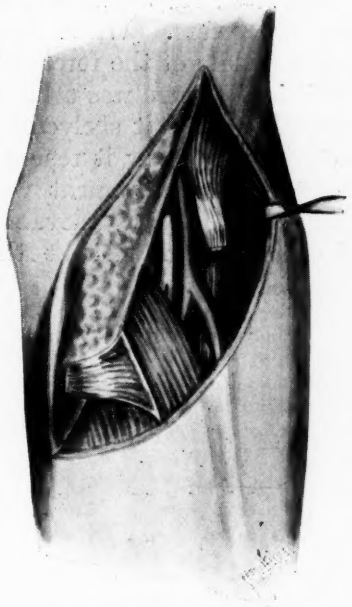

Fig. 155.—The approach to the part, with the lacertus fibrosa divided and attached distally.

Fig. 156.—Diagram of separation of the orbicular ligament.

Fig. 157.—Method of stitching of fascial implant.

of the forearm was not permitted till a day or two before leaving hospital a fortnight later, when he was sent home to be under supervision and not allowed to use the arm for any but restricted movement for one month.

The operation was performed on Feb. 14, 1930, and now, April 23, 1931, he has a history of being the 'hope of his side' at cricket, and has had one or two severe falls upon his injured arm without untoward result.

The lesson of the operation is, that for children at any rate the lacertus fibrosa is an efficient transplant, and it seems probable that the adult lacertus fibrosa (bicipital fascia) is strong enough for a like repair.

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[2] Cochrane, *Fractures and Dislocations*, 2nd ed.
[3] Thomas, T. T., *Ann. of Surg.*, 1929, lxxxix, 108.
[4] Scudder, *Treatment of Fractures*, 10th ed.
[5] Campbell, W. C., *Jour. Amer. Med. Assoc.*, 1929, xcii, 122.

THE SUCCESSFUL REMOVAL OF AN OSTEOMA OF THE FRONTAL SINUS.

By GEORGE ARMITAGE, Leeds.
ROCKEFELLER FELLOW, 1929–30.

With reference to my article on osteoma of the frontal sinus that appeared in the April number of the British Journal of Surgery, the following case may be of interest.

History.—Annie R., age 20, millhand, was admitted for a swelling in the frontal region which had been present for two years. When first noticed it was about the size of a marble, and in the middle of the forehead. It had increased gradually and painlessly until admission. There was a hard, nodular, fixed, and painless swelling in the mid-frontal region; it shelved off into the skull, but was more distinctly marked off above; below it reached to the supra-orbital ridges on both sides. The vertical measurement was 2½ in., the horizontal 3½ in. The skin over the tumour was freely movable. The patient suffered occasionally from slight headache, and thought the swelling might be due to a blow, but could not remember any.

Fig. 158.—Osteoma of the frontal sinus: anterior view. (*Specimen No. A.831, Pathological Museum, School of Medicine, the University of Leeds.*) (× 1.)

Operation (April 29, 1903—the late Mr. Edward Ward).—A frontal flap was turned down and the tumour exposed. It was found to be exceedingly hard, and its surface was tuberculated. The bone around it was chiselled away, and then by means of a Wheelhouse's elevator it was levered out. It was found attached by a small pedicle, which passed down into the right infundibulum. During removal the dura mater was slightly torn, and was sutured. Iodoform gauze was packed down into both frontal sinuses.

The patient was discharged well on May 16. *Fig.* 158 shows the specimen removed at operation.

REVIEWS AND NOTICES OF BOOKS.

Clio Medica Series. A series of Primers in the History of Medicine. Editor: E. B. KRUMBHAAR, M.D. F'cap 8vo. 1930 and 1931. New York: Paul B. Hoeber Inc. $1.50 per volume.

The Beginnings: Egypt and Syria. By WARREN R. DAWSON, F.R.S.E., Fellow of the Royal Society of Medicine, etc. Pp. 86 + x.

Medicine in the British Isles. By SIR D'ARCY POWER, K.B.E., F.R.C.S., Hon. Librarian at the Royal College of Surgeons of England, etc. Pp. 84 + x. Illustrated.

Anatomy. By GEORGE W. CORNER, M.D., Professor of Anatomy in the University of Rochester. Pp. 82 + xvii. Illustrated.

Internal Medicine. By Sir HUMPHRY ROLLESTON, Bart., G.C.V.O., K.C.B., M.D., Hon. D.Sc., D.C.L., LL.D., Regius Professor of Physic in the University of Cambridge. Pp. 92 + x.

Physiology. By JOHN F. FULTON, M.D., Stirling Professor of Physiology, Yale University. Pp. 141 + xvi. Illustrated.

PROFESSOR KRUMBHAAR did well when, as editor, he dedicated this series of Primers to Clio, one of the Muses. It was the duty of the nine virgin goddesses to bring to the mind of the poet the subjects of his song; it was their pleasure to confer upon him the gifts of music and grace. Clio in particular was the Muse of History, and the Greeks showed her with a scroll in her hands or sitting beside an open chest of books. Like Janus, she looked before and after, for with her eight sisters she had the gift of prophecy, and told of things to come as well as of things past.

The five primers of the series which have already appeared show that the Muse was indeed a woman—*varium et mutabile semper*—for the history of medicine has many facets. Each facet has been polished by the skilled hands of Mr. Warren Dawson, Sir D'Arcy Power, Sir Humphry Rolleston, Professor G. W. Corner of the University of Rochester, and Professor John F. Fulton of Yale. The series therefore should be read as a whole, for it is only by doing so that the beauty and interest of the story can be adequately appreciated. Each volume is clearly printed, is of a size suitable for the pocket, and is provided with an index. Each, too, is introductory to the subject of which it treats, and a bibliography is provided for those who wish to learn more. The volumes are published in New York by Paul B. Hoeber at a price of one dollar and a half, which is the equivalent in England of 6s. 6d. There are more illustrations in the later volumes than in the earlier ones, and in future volumes the illustrations might well be even more numerous. It is clear that Professor Krumbhaar has chosen the writer of each volume of the series with great care, and it is interesting, therefore, to see how each author has treated his subject in relation to the whole body of the history of medicine.

The first volume deals with the 'Beginnings of Medicine in Egypt and Assyria'. It is written by Mr. Warren R. Dawson, who has already published a larger work on the same subject under the title *Magician and Leech*. In the early times, about which he writes, Medicine was still allied to Magic, though he traces the history to a time in Egypt when specialism had become well established, to a time told of by Herodotus when "some physicians are for the eyes, others for the head, others for the teeth, others for the parts about the belly and others for internal disorders." Cyrus in those days sent to Egypt for a physician, believing that the Egyptians had the highest reputation for their skill in the healing art—an opinion endorsed by Homer.

Sir D'Arcy Power has limited himself to medicine in the British Isles, and shows how medicine and surgery in England have developed along lines which differ entirely

from other countries. Until lately the State has been unconnected with doctors and their affairs, which had gradually developed in the case of surgery from a religious trade guild into a profession. Relapses occurred from time to time, and advances were made by a few upright men at irregular intervals, but it is only in recent years that it has been possible to unite surgery with its sister branch of inner medicine. The story is well told, and Sir D'Arcy has added chapters on Nursing, on the Medical Societies, and on Some Masters of Medicine, taking John Arderne, William Harvey, Thomas Sydenham, William Smellie, William and John Hunter, Edward Jenner, Dr. Bright, Thomas Addison, Dr. Hodgkin, Sir James Y. Simpson, Sir William Stokes, and Lord Lister as examples.

Dr. George W. Corner has written a thoroughly reliable account of anatomy couched in so light a vein that it is eminently suitable for those who have but little medical or scientific knowledge. He has taken a wide view of the term 'anatomy' and has not confined himself either to anthropotomy or to comparative anatomy, but deals with histology and embryology. He devotes some wise words to current trends in anatomy, and if he is true to the principles enunciated, the University of Rochester will have cause to congratulate itself on having secured his services as Professor of Anatomy.

Sir Humphry Rolleston devotes a volume to a survey of the history of internal medicine, by which he means the old-fashioned term 'physic', and not the whole of the healing art. Beginning with Jewish, Hindu, and Chinese medicine, he tells of Greek medicine and of the manner in which it is linked with modern medicine. He then considers the Renaissance, which witnessed the revival of anatomy, physiology, and the application of chemistry and physics to the problems of medicine; and lastly he gives a most excellent account of the school at Leyden, famous for Hermann Boerhaave, 'the Dutch Hippocrates'. A chapter is devoted to the advances made in all directions during the last century, of which the present generation are the fruitful heirs.

The last volume of the series to appear is devoted to physiology, and is written by Dr. John P. Fulton, who was until recently a Fellow of Magdalen College, Oxford. It is designed perhaps more for medical students than for the general public, but it is well illustrated, is well written, and is easy of comprehension by those who only have the elements of physiology. Dr. Fulton begins by showing the influence of Aristotle and Galen which gave physiology a better start than was obtained by anatomy. He then proceeds to consider physiology under the headings of the vascular, the respiratory, and the digestive systems, with a chapter on the physiology of the nineteenth century and an excellent account of the rise and development of the teaching laboratories. The epilogue on the trend of modern physiology is of especial value, and should be read and reflected upon by all who are interested in the subject of medical education and of the part in it played by the ancillary sciences.

In conclusion, the publishers are to be congratulated on the enterprise which has led them to launch the series, which is likely to become a success commercially. They announce twelve more volumes on the same lines as being in preparation, and promise other volumes in addition.

Sir D'Arcy Power: Selected Writings, 1877–1930. Medium 8vo. Pp. 368 + x, Illustrated. 1931. Oxford: The Clarendon Press. 25s. net.

THIS volume, forming an anthology of his writings, was presented to Sir D'Arcy Power by a number of his friends to celebrate his seventy-fifth birthday, Nov. 11, 1930. It contains three surgical and thirteen historical papers out of more than six hundred articles and volumes, which have proceeded from his pen. We are sure that most readers of this fascinating volume will want to see more of the remaining five hundred and ninety. For besides his eminence as a surgeon there is no one who has made the history of medicine in England in the past so thoroughly his own as Sir D'Arcy, no one who can make Harvey, Hunter, or Arderne so live again before us, while he explains their hopes and work by details which he has gathered from a

thousand sources during fifty years of ceaseless study and writing. Nothing startles the casual reader more than his minute knowledge of very minor actors who may be mentioned, for instance, in Ward's *Diary*.

The pathos, too, of many of his pictures is very great. For instance, take his story of Hunter, that "fierce rude prophet", crippled and hindered in his life's work not only by his poor education and the imperfect science of the time, but still more by the terrible anginal and cerebral attacks, the result of the syphilitic inoculation which he foolishly tried on himself twenty-six years before his death.

One of the most pathetic stories in the English language is that of poor Pepys detailing month by month the torments he suffered in his work from bad eyestrain, probably hypermetropic astigmatism, and his final discourse with himself on the necessity of at once stopping his beloved, his incomparable, *Diary*. This was thirty-four years before his death. What a brilliant picture of the Revolution we have lost by this stoppage! In our author's hands the accounts of Pepys' constant breakdowns and his efforts to find some kind of glass to relieve him become a great tragedy the sadness of which is increased by the reflexion that, though scientific treatment was then unknown, relief might probably have been had at once if anything had led him to read through a slit while using his glasses.

If anyone thinks that medical history is void of tragic interest, let him read Sir D'Arcy's vision of the deaths of Wallace, Wat Tyler, and the martyrs at the gate of St. Bartholomew's, or even the story of poor Dr. Harvey in his sixtieth year, riding forty miles a day on a mission in Italy and then imprisoned in a filthy lazaretto and attacked by sciatica, as he details his woes in letters to Lord Feilding and implores his help for release.

In these selected papers we have an outline of the story of surgery in England from 1300 to 1850 with charming sketches of many of the actors. The author begins with his hero Arderne, and the *De Arte physicali* which he has since translated and edited. But we must go to the Early English Text Society for his delightful biography of Arderne and other translations of his works.

In discussing the various classes of mediæval medical men he touches on the difficult question as to what surgeons had the title of 'Magister', suggesting that, besides University graduates, all members of the guild of surgeons enjoyed it, but we find this very guild appointing four of their members a year to be Magistri or overseers. Still it is possible that the suggestion is correct, and when late on the absurd title 'Master' Doctor Harvey turns up, we have to confess almost anything seems possible.

Sir D'Arcy remarks on the unique use of the word 'furlong,' (p. 44), as a measure of time. We would suggest that it is like the 'time of a Paternoster' given in sundry receipts. Indeed, the Oxford Dictionary, quoting a like phrase from the *Miller's Tale*, says: "They sitten stille wel a furlong way", and in *Two Cookery Bookes* we read, "Let them stonde a furlong way or two." On p. 60 he quotes another archaic phrase, "a minute of an hour", i.e., 60 seconds.

Again, while the statement on p. 45 that hospitals had no separate medical staff before the sixteenth century may be true in a sense, it is clear that physicians were wardens in some cases—for example, Dr. Pascal, of Bonona, was Master at Preston in 1265, and Dr. Recouchez at Pontefract Hospital. Indeed, at York there was Ann Medica as a sister, and of course in monasteries there was always an official Infirmarius.

It is interesting to learn that the best mediæval surgeons were often itinerant, living with the patient till he was cured, and paid partly in cash and partly by an annuity. The description of the education of a surgeon under Vicary and his successors is excellent, but we wish that more emphasis had been put on the fact that much of the system existed earlier than 1540; thus the Royal Charter of 1462 confirms to the Company of Barbers and Surgeons the right of examining surgeons, and their Ordinances in 1529 enforce attendance on the weekly lecture on surgery for those on the surgical side.

In his account of Harvey, Sir D'Arcy suggests as a possible cause of Harvey's coming to St. Bartholomew's his friendship with Inigo Jones, who was born in the

Hospital precincts and was studying in Italy at the same time that Harvey was there. Whether that is so or not, young Harvey after his return married the daughter of Dr. Lancelot Brown, and in 1609 got a royal recommendation and was appointed Physician. His service there lasted some forty-three years, but in the last twelve he was often compelled to be absent either in personal attendance on the king, or when sent on a royal mission. The mission in 1636 to Vienna and Italy under the Earl of Arundel makes an extraordinary story, the details of which were first discovered by Sir D'Arcy. He shows us the embassy making its way through the horrors and devastation of the Thirty Years War, the crowds dying of starvation whom they had to relieve, and the freebooters whom they had to fight at every turn. At last they reached Vienna and Prague, and had audiences with the Emperor, but Harvey was sent on alone to Venice about some pictures for the king. It was on this four-hundred mile ride that he was imprisoned at Treviso, but he finally carried out his mission, and after a visit to Florence and Rome rejoined his party in safety. The whole forms a striking episode in Harvey's life and a good picture of travel at the time. It is curious to compare with it Nicholas Ferrar's journey over much the same ground twenty years earlier and his detention in a lazaretto.

The paper on the first part of John Ward's *Diary* is of very great interest as a picture of the lives and work of Willis, Lower, and other scientific physiologists just before the foundation of the Royal Society, an epoch more fruitful and important in English medicine than any other till the nineteenth century. The author shows that Ward actually records Joliffe's discovery of the lacteals a year or so before any one else published a claim.

The Bradshaw Lecture on cancer of the tongue was well worthy of inclusion in this volume. It brings forward so many facts which are apt to be overlooked, and attains to some curious results. The lecturer made a remarkable search in ancient and mediæval literature to find out in what degree the disease was prevalent in the past. To his surprise he could not find a certain instance of it before the case given by Alex. Read in 1635. Since then there has been a huge increase in male patients, while in women and animals it remains comparatively rare as before. After reviewing many statistics on this disease and on the co-existence of syphilis and dental caries, he concludes that a great predisposing cause of the increase has been old and inadequately treated syphilis and alcohol. The exciting cause is usually irritation from tobacco and in a lesser degree from dental caries, but none of these things will produce it apart from the presence of the unknown specific cause.

The history of the Royal College of Surgeons of England will appeal to everyone with its amusing accounts of the lectures and the fate of Hunter's Museum, Smollett's description of an examination, and its many stories of Abernethy, Blicke, Sheldon, and South.

A Text-book of the Surgical Dyspepsias. By A. J. WALTON, M.S., M.B., B.Sc. (Lond), F.R.C.S., Surgeon, London Hospital; Late Hunterian Professor, Royal College of Surgeons. Second edition. Demy 8vo. Pp. 720 + viii, with 286 illustrations in the text and 2 plates. 1930. London: Edward Arnold & Co. 42s. net.

THE second edition of this book presents a clear account of the practice of the author and his views on those conditions which give rise to upper abdominal discomfort. As is to be expected, definite and almost dogmatic views are expressed in relation to the conditions discussed, and the author's reasons for holding them are given. We can see and follow his line of thought in every instance, although we may not always be in complete agreement with him. Special mention must be made of the chapter on the difficult subject of visceroptosis. The clinical picture is admirably drawn, the indications for operation are carefully considered, and the conclusion, with which most of us will agree, is reached that operations are, as a rule, to be avoided. It is perhaps unfortunate that there is no mention of operations on the abdominal sympathetic, but possibly it is not considered that their results are as yet sufficiently uniform for them to be included in a book of this kind.

In the chapter on gall-stones it is recommended that cases of acute cholecystitis should be operated on without delay. Certainly a well reasoned justification is

given, but we are inclined to think that the dangers of delay are rather exaggerated, and that the fact that the normal course for cases allowed to settle down is tranquil and not attended by complications is not sufficiently brought out. It may be sound theoretical reasoning to compare the gall-bladder with the appendix, but practical experience shows us that the two viscera do not behave in the same way when inflamed. Exploration of the common bile-duct from within is advocated in every operation on the biliary system. We do not agree with this, as there is no doubt that careful palpation from without, rolling the duct between the finger and thumb, will reveal not only a stone but even grit to an observer who has trained himself by always examining the duct in this way.

The chapters on the stomach and duodenum are admirable; they are lucid and complete, and it is only in details that we can offer any criticism. For example, in the description of the operation of gastro-enterostomy, it is stated, "the excess of mucosa is not removed as this step appears to increase the likelihood of a gastro-jejunal ulcer." That is a very important statement, and is contrary to the experience of many surgeons. To give it without any justification or explanation is not enough to make a surgeon who does not believe it even consider it. In discussing perforated ulcers it is said that the pain may be sufficiently severe to cause the patient to writhe about. We cannot accept this. The typical picture is one in which the patient lies absolutely still, in which the breathing is entirely thoracic, and in which it appears as if he is afraid that the slightest movement might cause an exacerbation of his suffering. We are very much in agreement with the opinion which is voiced that operation should be undertaken at the earliest possible moment. To wait for an improvement after the collapse which is seen in the first hour or two is to wait for something which will not occur. The condition of the patient and the prognosis become worse with each succeeding hour. In the treatment of a perforated ulcer it is said that the fluid which has escaped should be mopped out and that when drainage has been instituted the pelvic tube may be left in position for ten days. If it is recognized that this mopping out must only be done in the mildest of ways and that perhaps any solid matter which is seen may be removed, then it is harmless. To attempt to clean out the fluid present in anything like a methodical manner is to increase the shock of the operation by a procedure which is unjustifiable and unnecessary. The time the tube is left in position is a matter of opinion, but we cannot agree that by rotating it each day the danger of ulceration into the bowel can be avoided.

Finally, we cannot agree with the author in his treatment of the appendix during the course of an abdominal operation for some other condition. He states that he finds evidence of disease in only 4 per cent of cases and that he only removes the appendix in those cases. An appendicectomy does not appreciably prolong the time taken by the operation, and it is a safeguard for evermore against the development of an appendicitis. It has been truly said that the only disadvantage is that it derives some surgeon of a fee at a future date.

This is a book which represents very clearly the surgical view in cases of dyspepsia; it embodies the teaching and practice of the large majority of surgeons who are engaged in this kind of work in a way in which no other text-book with which we are familiar does. If it is a little individual in parts, that is only what we can expect from an author with the skill and experience of Mr. Walton. The publishers are to be congratulated on the clearness of the text and on the way in which they have reproduced the large number of really useful illustrations.

Diseases of the Tongue. By WALTER G. SPENCER, M.S., F.R.C.S., Consulting Surgeon, Westminster Hospital; and STANFORD CADE, F.R.C.S., Assistant Surgeon, Westminster Hospital. Being the third edition of Butlin's *Diseases of the Tongue*. Demy 8vo. Pp. 561 + xvi, with 123 illustrations in the text and 20 coloured plates. 1931. London: H. K. Lewis & Co. Ltd. 35s. net.

THE success of radiotherapy in buccal cancer has led to a revival of interest in diseases of the tongue, and for a monograph on such a subject there could be no

better foundation than Butlin's classic. In this third edition the arrangement of the subject matter follows fairly closely that of the second edition (1900), but the work has been entirely rewritten, so that we cannot but regret the loss of Butlin's easy style. Chapters on the anatomy and physiology of the mouth are followed by a full account of every known disease of the tongue, and there are also chapters on the salivary glands and thyroglossal tract. Rather less than half the book is devoted to malignant disease of the tongue, and some fifty pages of this section are occupied by case reports. Radiotherapy of carcinoma of the tongue is most fully described. The stress laid upon the factors essential for success, the clear account of the clinical changes seen in an efficient reaction, and of the means by which errors may be avoided or corrected, are especially noteworthy. The value of such attention to detail is emphasized by the authors' results—healing of the primary growth being obtained in 74 per cent of the cases (cf. the Paris figure of 46 per cent).

With regard to the lymphatic glands of the neck, stress is laid upon the absolute necessity of treating them in every case, however early, by radium needles inserted through the skin for unpalpable glands; by block dissection for operable palpable glands; and by radium insertion by open operation for inoperable glands. The authors are hopeful that the results of radium needling by open operation may soon prove as good as those of block dissection in the palpable and operable group of cases.

A knowledge of the detailed histology of an epithelioma is shown to be of value in some cases, the anaplastic type of growth and the lympho-epithelioma both being very radiosensitive. In a monograph of this kind a fuller account of the detailed histology in the light of Broder's work might have been included, and this is the more surprising in view of the authors' unequivocal statement that a biopsy does no harm.

The book is profusely illustrated, and many of the coloured plates have been redrawn from the second edition by Mr. Sewell—unfortunately they have now lost that life-like appearance which was one of the distinguishing features of Butlin's book. The bibliography is complete and excellently arranged, and the book is one which should appeal not only to the radium expert, but to every general practitioner who is in the habit of saying to his patients 'put out your tongue'.

Deep X-ray Therapy in Malignant Disease. A Report of an Investigation carried out from 1924–1929 under the direction of the St. Bartholomew's Hospital Cancer Research Committee. By WALTER M. LEVITT, M.B., D.M.R.E., Medical Officer in Charge of the Radiotherapeutic Research Department. With an Introduction by SIR THOMAS HORDER, Bart., K.C.V.O., M.D., F.R.C.P., Chairman of the Cancer Research Committee. Post 8vo. Pp. 128 + xiv, with diagrams. 1930. London: John Murray. 10s. 6d. net.

THE book is published by the committee of the *St. Bartholomew's Hospital Reports* as a supplementary volume. The author has given in his Report to the Cancer Research Committee of the Hospital a fair and unbiased account of his work as medical officer in charge of the Radiotherapeutic Research Department during the five years 1924–9. During that period some 200 cases of malignant disease were treated by irradiation, the majority by X rays, but in a few cases a combined treatment by X rays and radium was carried out.

The book is divided into three sections which deal with apparatus, technique, management of cases during and after treatment, results of treatment, and so on. Elaborate statistical tables are given, but the numbers are of course too small for comparison with the available statistics from other centres. There is little that is new to record, but attention is drawn to the six-field method of Finzi, which was used in treating malignant disease of the 'upper air-passages' and which seems to have given good results. It should be noted that nearly all the cases treated were inoperable, and when this is borne in mind the results obtained are just what one would expect—a temporary alleviation of distressing symptoms, and in some cases, so far as could be ascertained clinically, a temporary disappearance of the growth. The majority of the patients died within the first two years after treatment.

The book can be recommended to radiotherapists who wish to compare the methods used and the results obtained with their own. They will find them very much alike.

Radium Therapy: Principles and Practice. By G. E. BIRKETT, *M.C.*, B.A. (Cantab.), M.R.C.S., Hon. Radiologist, Manchester and District Radium Institute and Christie Hospital for Cancer. Royal 8vo. Pp. 186 + x, with 6 colour and 13 half-tone plates, and 52 illustrations in the text. 1931. London: Cassell & Co. Ltd. 17s. 6d. net.

IN this book is gathered much of the experience gained by Dr. G. E. Birkett during a number of years' work at the Manchester and District Radium Institute.

It is with great regret that we have to notice that Dr. Birkett died shortly before its publication. Seriously wounded and permanently crippled in the Great War through a gunshot wound, he took up radium work with great enthusiasm and achieved much success. He was prominent in leading the way in treating carcinoma of the tongue, and it was his work that helped to convince many surgeons of the usefulness of this method of treatment. We deplore his loss, but are glad that he lived long enough to place on record some of the most important of his observations.

The book is a very useful addition to the researches in radium therapy, and not the least useful is the first chapter on General Principles of Radium Therapy.

Annals of Roentgenology: a Series of Monographic Atlases. Edited by JAMES T. CASE, M.D., Professor of Roentgenology, Northwestern University Medical College, Chicago. Volume XII. The Chest in Children. By E. GORDON STOLOFF, M.D. Crown 4to. Pp. 432 + xxx, with 401 illustrations. 1931. New York: Paul B. Hoeber Inc. $15.00.

THIS book is arranged as a classification of the diseases of the chest in children, which is illustrated whenever possible with skiagrams. It is essentially a record of the author's experience of the evidence afforded by X rays of the various disease processes. The collection of so much material is a great feat, for each example shown must be the result of selection from a vastly larger store. The skiagrams are admirably produced. One regrets that there is not more about the healthy chest. Whether or not a shadow comes within the range of the normal is so often a matter on which one needs help. There is no special section on heart shadows, an omission which seems strange when even the thyroid has its own chapter. The book is most helpful in consolidating one's impressions of the X-ray appearances of conditions with which one is familiar. It is doubtful whether it can give help in diagnosing a difficult case.

Fractures and their Complications. By GEORGE EWART WILSON, M.B. (Tor.), F.R.C.S., F.A.C.S., Surgeon-in-Chief, St. Michael's Hospital, Toronto, etc. Medium 8vo. Pp. 415 + viii, with 306 illustrations. 1931. London: Ballière, Tindall & Cox. 35s. net.

THIS most recent addition to the many works on fractures has a very distinct character of its own—in fact it is a representation of the author's very practical methods and not an attempt to present any other than these. The two main elements in the technique advised are: the routine use of the fluoroscopic screen both for diagnosis and treatment, and the employment of plaster-of-Paris as a fixation agent almost to the exclusion of everything else.

This rather narrow view of the subject of fractures is open to criticism both in principle and detail, and frankly we do not believe that this confinement of our resources to so limited a choice of methods is an advance. But at the same time it is helpful to have this treatise for the sake of reference, to see what the out-and-out plaster enthusiast claims to be able to do in any special condition. The routine use of the X-ray screen for treatment is certainly a most attractive idea, but it seems to us unreasonable to suggest that it is necessary in all cases. The fact that many fractures, especially of the forearm, will make a much quicker functional recovery if not fixed in plaster for several weeks is ignored. The book is a clear exposition of a special method, and other methods are hardly mentioned.

A consideration of fractures of the skull and spine is included in the book, though these represent such totally different problems from fractures of the long bones. Operative treatment for fractured spine is relegated to a very minor place, and the

value of immediate stabilization operations for cases of compression fractures or other fractures of the spine without cord lesions is not mentioned.

When the author does leave his narrow plaster pathway to describe operative methods, we think that he is confused partly because he uses complicated devices. For example, the open operation for comminuted fractures of the humerus is difficult either to understand or believe in. Fractured olecranon has to be treated by an axial screw—which, of course, will take no hold on the loose cancellous tissue of the ulnar shaft—then the limb is to be put up in plaster for four weeks! If an open operation is properly done on the olecranon, no splint is required and the patient should be back at work in four weeks. The same kind of criticism applies to the operation for fractured patella, which has to be 'plastered' for five weeks.

We think it deplorable that a modern book on fractures should devote so much attention to adhesive traction appliances for leg fractures and so little to transfixion pins or the taut wire traction. We do not agree that it is never necessary to do an open operation for fractures of the tibia and fibula. As a clear exposition of an excellent method the book is good. As a general exposition of the treatment of fractures it is too limited to be of any real value.

Cancer and Race: a Study of the Incidence of Cancer among Jews. By MAURICE SORSBY, M.D., F.R.C.S.E., Hon. Assistant Surgeon, Ear, Nose, and Throat Department, London Jewish Hospital, etc. With a Preface by Lieut.-Col. F. E. FREMANTLE, M.A., M.D., M.Ch., F.R.C.P., F.R.C.S., D.P.H., M.P., Consulting County Medical Officer of Health for Hertfordshire. Conducted under the auspices of the Jewish Health Organization of Great Britain. Demy 8vo. Pp. 120 + xvi. 1931. London: John Bale, Sons & Danielsson, Ltd. 7s. 6d. net.

THIS is an interesting short study designed to throw light on the problem of the racial factor in the causation of malignant disease. For a long time there existed a vague and non-substantiated idea that Jews were immune, or at any rate less liable to cancer than non-Jews. This question has been settled once and for all by the writer. He shows that Jews as a race neither enjoy an immunity nor suffer from a predisposition to cancer, and that as communities they show vagaries towards a higher or lower incidence of cancer which are similar to the larger communities in which they reside.

The author, however, calls attention to the variation of the incidence of cancer of certain organs in Jews. He gives evidence for his belief that the incidence of cancer of the penis, the tongue, and buccal cavity is lower in Jews than in non-Jews, whereas the incidence of cancer of the intestinal tract is higher. He attributes the low incidence of cancer of the penis, which is shared by Mohammedans, to circumcision, and not to any racial immunity. The low rate of cancer of the tongue he believes is connected with the low rate of syphilis among Jews, and the low rate of cancer of the uterus to the beneficial rules of sexual hygiene which were imposed by the Mosaic Law. No explanation for the high incidence of intestinal cancer is given. The conclusion of the argument appears to be that, in the incidence of cancer, Jews do not show any specific racial characteristic.

Le Cancer. By G. ROUSSY. Second edition. 1929. Pp. 846, with 280 illustrations. 1929. Paris: Masson et Cie. Fr. 100.

THIS book forms the second volume of the great system of medicine published under the auspices of Roger, Widal, and Teissier. The work represents one of the most complete discussions of our present knowledge of tumours available in any language. Like so many French books, it suffers from the grave defect of possessing no index, is printed on poor paper, and bound in a peculiarly fragile manner. Fortunately, many of the pictures are inset on good art paper and cannot fail to be of great value. The book starts with a short introduction to the history of our knowledge of cancer; then a long chapter deals with those agents known to be capable of producing malignant tumours, and there is here an excellent account of the various forms of cancer associated with industrial processes.

The section on pathogenesis is short, and contains little more than a résumé of the various hypotheses that have been published everywhere. Almost 250 pages are devoted to the pathological anatomy and histology of malignant tumours, and here many excellent photomicrographs and drawings have been included. The English reader may find some difficulty in this section because of the terminology used in France. The custom of referring to almost all forms of carcinoma as epithelioma is confusing at first, but this is only a temporary difficulty. A particularly good part of this chapter is the description of tumours affecting the nervous system. About 150 pages are devoted to a discussion of the pathological physiology of the cancer process, and there probably exists no better description of the natural history of the disease.

Malignant tumours as they occur in the lower animals are briefly described, and there is an excellent description of the experimental production of cancer. A short but interesting section is that devoted to the tumours occurring in plants. The general symptomatology of malignant tumours is described in detail, as are various diagnostic methods. Only about 150 pages are devoted to the discussion of treatment. Individual surgical procedures are not described, but radiological therapy is discussed at some length.

The last seventy pages of the book are of great interest as giving statistical information of the mortality from cancer in most of the civilized countries. In the last few pages the author describes the steps being taken in different countries to enlighten the general public as to the nature and prevention of malignant tumours.

The whole book is a monument to the industry of Professor Roussy and his two collaborators, Dr. Roger Leroux and Dr. Maurice Wolf.

Physiologie pathologique chirurgicale. Inflammations, Effects des Traumatismes, Réparation des Plaies, Greffes, Maladies des Os, des Articulations, des Vaisseaux et des Nerfs. By R. LERICHE, Professeur de Clinique chirurgicale à la Faculté de Strasbourg; and A. POLICARD, Professeur d'Histologie à la Faculté de Lyon. Medium 8vo. Pp. 212. 1930. Paris: Masson et Cie. Fr. 26.

THIS little volume, in common with many another work associated with Leriche, will be found to be not only of interest to those who are trying to correlate recent advances in physiology with the problems of surgery, but also a thought-provoking stimulus to everyone who is attempting to understand the nature of disease as a whole. It is not merely a collection of new facts; it is more a criticism of commonly accepted explanations of certain phenomena in the light of new knowledge, and the statement of a fresh point of view. There is scarcely a page in the book that does not call for an exercise of the critical faculty, and time and again the reader is overcome either with the delight of seeing some well-known phenomenon in a truer light, or with a burning desire to argue with the author. It is the product of a fertile mind approaching old problems with a vision unclouded by preconceived ideas, and is of value not only for the thoughts expressed therein, but for the ideas for further research which a critical reader may obtain from its study.

An attempt is made to elucidate the nature of the process of disease; and in the case of diseases of unknown cause it is hoped that by studying their effects and expressing the particular tissue reactions in terms of physiology as at present understood, the cause may eventually be discovered. As the authors state in their introduction, the volume is intended to encourage the clinician to attack his problems from the standpoint of the experimental pathologist, and the pathologist to study disease while function alone is affected and before the stage of structural change has been reached.

Seeing that function is given the most prominent place, it is natural that the importance of tissue nutrition should be stressed, and this gives Leriche the opportunity to bring forward his favourite subject of vasomotor control. His statement, "It may be said that our physical and psychic existence is nothing but a series of vasomotor reactions" may be regarded as the text and keynote of the whole work; yet one cannot help noting the remarkable success which he records following

operative procedures designed to interrupt the paths along which he believes the controlling impulses to travel.

In a work of such a character it is difficult to single out any chapters for special praise. Those dealing with inflammation and arthritis are excellent, but the most striking are the sections on bone—especially the mechanism underlying the absorption and deposition of bone in pathological states—and on the arteries and veins. The way in which changes in the blood-vessels are linked up with clinical phenomena, and more especially with sensations of pain, is most remarkable. And most astonishing of all are certain of the methods recommended for the relief of such pain—methods the value of which is vouched for by the authors after fifteen years of careful research.

This book is to be heartily commended, not because it is startling and unorthodox and entertaining, though it may be considered to possess all those attributes, but because it will be illuminating and give food for thought to all who are interested in advancing the science of surgery.

Surgical Diagnosis. By American authors. Edited by EVARTS AMBROSE GRAHAM, A.B., M.D., Bixby Professor of Surgery, School of Medicine, Washington University, St. Louis, etc. Volume III. Large 8vo. Pp. 1043, with 446 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. Per set—three volumes and index volume—£7 10s.

THE first two volumes of *Surgical Diagnosis* were reviewed in Vol. XVIII, No. 69, of this JOURNAL. Volume III is now presented, together with a separate Desk Index Volume, which is a general index to the three volumes. It deals with the surgery of the thorax, breast, liver, pancreas, rectum, genito-urinary organs, and nervous system. The remarks made previously concerning the first two apply equally to this volume. It is a useful work of reference for the general practitioner who wishes to steer his patient through difficult times.

The Metabolism of Tumours. Investigations from the Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Edited by OTTO WARBURG, Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Translated from the German edition, with accounts of additional recent researches, by FRANK DICKENS, M.A., Ph.D., whole-time worker for the Medical Research Council, Courtauld Institute of Biochemistry, Middlesex Hospital, London. Demy 8vo. Pp. 327 + xxix. Illustrated. 1930. London: Constable & Co. Ltd. 40s. net.

THE work of Warburg and his school on the respiratory processes in living cells, and especially in those of tumours, is too well known to need discussion. Much of this work is at present too theoretical to be of immediate interest to the practising surgeon, but to the research worker on cancer it is of very great interest and importance. This translation of the German edition of Warburg's collected papers, together with a number of recent additions, will be heartily welcomed by all workers concerned in cancer research.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI Fourth Series), No. 1. January, 1931. Royal 8vo. Pp. 126. 1931. London: The Lancet Ltd. Annual subscription £2 2s. net; 12s. 6d. per issue.

THIS number contains a symposium on streptococcal infections, which is similar to a symposium on staphylococcal infections appearing in the number for April, 1930.

Dr. Ryle deals with the natural history, prognosis, and treatment of streptococcal fever in an article notable for its restraint and sound common sense. Perhaps the most valuable part of his article is the review of the various forms of treatment which have been keenly advocated by enthusiasts. Of serum therapy he says: "On the whole, we must confess that there seems very little evidence in favour of the efficacy of serum therapy. It is very generally employed, and had its effects been in any way dramatic there must by now have been a stronger consensus of opinion to support it." Again, his conclusions concerning intravenous medication

are worth quoting: "Mercurochrome and other chemical antiseptics by the intravenous route are to be avoided. There is no sound evidence, whether experimental or clinical, that they do good. Some of them have undoubted toxic effects. Their administration can be distressing to the patient and they are capable of causing disturbing symptoms."

G. F. Gibberd writes the article on streptococcal puerperal sepsis, and it is interesting to note that he too looks with disfavour on methods designed to kill the streptococci in the blood-stream. He discusses the place of blood transfusion in puerperal streptococcal infection, and defines it as follows: invaluable as a prophylactic measure after hæmorrhage, and as a therapeutic measure nearly always good, provided the reaction does not kill the patient.

Dr. Mollison describes the streptococcal infections of the throat. Mr. Layton describes hæmolytic streptococci in the mastoid, and Dr. Barber those of the skin, while Dr. Knott deals with the streptococci from the standpoint of the bacteriologist.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Beobachtungen am gebrochenen Knochen. By Dr. C. BAUER (Köln a. Rh.). Würzburger Abhandlungen Bd. 26, H. 12. Super royal 8vo. Pp. 432–448, with 12 illustrations. 1931. Leipzig: Johann Ambrosius Barth. RM. 1.60.

The Veterinary Journal. Edited by FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E., Hon. Veterinary Surgeon to H.M. the King. Special surgical number. Royal 8vo. Pp. 107–158. Illustrated. March, 1931. London: Baillière, Tindall & Cox. 2s. net.

Recent Advances in Radiology. By PETER KERLEY, M.B., B.Ch. (N.U.I.), D.M.R.E. (Camb.), Assistant Radiologist, Westminster Hospital. Demy 8vo. Pp. 324 + viii, with 120 illustrations. 1931. London: J. & A. Churchill. 12s. 6d. net.

Crippled Children: their Treatment and Orthopedic Nursing. By EARL D. MCBRIDE, B.S., M.D., F.A.C.S., Instructor in Orthopedic Surgery, University of Oklahama Medical School. Large 8vo. Pp. 280, with 159 illustrations. 1931. London: Henry Kimpton. 15s. net.

Verlauf der wichtigsten Knochen- und Gelenkerkrankungen im Röntgenbilde. Eine anschauliche Prognostik. By Privatdozent Dr. med. VICTOR HOFFMANN, Oberarzt der chirurgischen Universitätsklinik im Augusta-Hospital zu Köln. English and German text. Imperial 8vo. Pp. 264 + x, with 584 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 66; bound, RM. 69.80.

Die Bakteriologie der Wurmfortsatzentzündung und der appendikulären Peritonitis. By W. LOHR (Magdeburg) and L. RASSFELD (Altona). Royal 8vo. Pp. 95 + viii, with 46 illustrations. 1931. Leipzig: Georg Thieme. RM. 12.

Röntgendiagnostik der Gallenblase. By Priv.-Doz. Dr. F. EISLER and Dr. G. KOPSTEIN. Radiologische praktika Bd. XVII. Super royal 8vo. Pp. 153 + viii, with 151 illustrations. 1931. Lepzig: Georg Thieme. Bound, RM. 18.60.

Operative Chirurgie der Knochenbrüche. By Professor FRITZ KÖNIG (Würzburg). Vol. I. Operationen am frischen und verschleppten Knochenbruch. Royal 8vo. Pp. 194 + vi, with 200 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 27; bound, RM. 29.80.

Die Lebensvorgänge im normalen Knorpel und seine Wucherung bei Akromegalie. By J. ERDHEIM, A.O. Professor an der Universität Wien. Pathologie und Klinik in Einzeldarstellungen, Bd. III. Large 8vo. Pp. 160 + viii, with 31 illustrations. 1931. Berlin and Vienna: Julius Springer. Paper covers, RM. 18; bound, RM. 19.60.

Collected Papers 1904–1929. By EDWIN BEER, M.D. (New York). Large 8vo. Pp. 827 + xii, with 252 illustrations. 1931. New York: Paul B. Hoeber Inc. $7.50.

Thomson and Miles' Manual of Surgery. By ALEXANDER MILES, M.D., LL.D., F.R.C.S. (Ed.), Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, M.D., F.R.C.S. (Ed. and Eng.), Professor of Surgery, University of Edinburgh. Vol. I. General Surgery. Eighth edition. Crown 8vo. Pp. 574 + xvi, with 176 illustrations. 1931. London: Oxford University Press. 12s. 6d. net.

Das Pollersche Verfahren zum Abformen an Lebenden und Toten sowie an Gegenständen. By ALPHONS POLLER. Edited by E. B. POLLER and E. FETSCHER. With a Foreword by Prof. Dr. C. VON ECONOMO (Vienna). Large 8vo. Pp. 216 + xii with 129 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 12; bound, RM. 14.

Cancer: International Contributions to the Study of Cancer. In honour of James Ewing. Edited by FRANK E. ADAIR, M.D., F.A.C.S., Attending Surgeon to the Memorial Hospital, New York. Super royal 8vo. Pp. 484 + xx, with 168 illustrations. 1931. London: J. B. Lippincott Co. 45s. net.

Surgery: its Principles and Practice. For Students and Practitioners. By ASTLEY P. C. ASHHURST, A.B., M.D., F.A.C.S., Professor of Clinical Surgery in the University of Pennsylvania. Fourth edition, thoroughly revised. Large 8vo. Pp. 1189 + xii, with 1063 illustrations and 15 coloured plates. 1931. London: Henry Kimpton. 45s. net.

La Chirurgia della Innervazione periferica del Simpatico (Chirurgia del Dolore). By Prof. IGNAZIO SCALONE (Milan). Large 8vo. Pp. 254 + xvi, with 91 illustrations. 1931. Milan: Ulrico Hoepli. L. 30.

Abdominal Pain. By JOHN MORLEY, Ch.M., F.R.C.S., Assistant Surgeon, Manchester Royal Infirmary, etc. With an Introduction by J. S. B. STOPFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Medium 8vo. Pp. 191 + xvi, with 22 illustrations. 1931. Edinburgh: E. & S. Livingstone. 10s. 6d. net.

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